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THE *JOURNAL OF PERIODONTOLOGY* is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society. The program for its 1941 annual meeting is in this issue. Abstracts of important articles on periodontia that appeared during the year of 1940 are published in this number.

We are grateful to all who have made available the subject matter from which the abstracts and copies of abstracts have been made, including the authors of the original articles, owners of the journals mentioned in the text of the abstract section, and those who have aided in any way in compiling the material comprising this issue.

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"The progress of chronic and acute inflammation of the gingiva into the supporting structures was studied histologically in jaws ranging from 11 to 74 years which were secured at autopsy.

"A review of the literature revealed different opinions in regard to how inflammation of the gingivae extends into the supporting structures.

"The histological studies revealed the following findings: Chronic inflammation of the gingiva progresses, following the course of the blood vessels, into the bone marrow spaces and on the periosteal side of the alveolar bone; only in exceptional instances does it penetrate into the periodontal membrane. Resorption of the alveolar crest from the gingival side leads to destruction, first, of the supporting alveolar bone and then the lamina dura. The fatty marrow is replaced by fibrous marrow.

"The distribution of the blood vessels explains the pathological picture. The effect of gingivitis on the supporting structures is interpreted as osteitis." *Summary*—"Progress of Gingival Inflammation into the supporting Structures of the Teeth," Joseph P. Weinmann, M. D., Page 81.

THE JOURNAL OF PERIODONTOLOGY

JULY, NINETEEN HUNDRED FORTY-ONE

Progress of Gingival Inflammation into the Supporting Structures of the Teeth*[1]

JOSEPH P. WEINMANN, M. D., *Chicago, Illinois*

IT is a matter of fact that the terminology of the disease group attacking the supporting structures of the teeth is in a state of great confusion. Single investigators and groups of clinicians have made and are making attempts to clarify the field and come to a general understanding. It is obvious that a clear idea of the pathological process is required before designating the disease by a term which expresses the essential nature of that particular disease entity.

This situation induced histological investigations, the purpose of which was to answer the question as to where and how the inflammation of the gingivæ progresses into the supporting structures of the teeth.

In limiting myself to a circumscribed analysis no attempt is made to solve the problem of terminology. A report of the findings seems to be justified by the fact that a review of the literature revealed controversial answers to the problem in question.

REVIEW OF THE LITERATURE

The answers to the question, where and how does inflammation of the gingiva progress into the supporting tooth structures, can be divided into different groups.

One group of investigators claims that inflammation of the gingivæ proceeds into the periodontal membrane. I am referring to G. V. Black,[1] E. S. Talbot;[13] E. W. Fish[4] and A. H. Merritt.[9] Their conception is indicated by terms such as, "chronic suppurative pericementitis," or, "periodontitis." Black defined "chronic suppurative pericementitis" as a "chronic infection of the supporting structures of the teeth." He considered the reason for the extension of gingivitis into the supporting bone structures to be that "the lymphatics within the periodontal membrane form the natural route for the movement of infection. These vessels accompany the blood vessels *from the gingivæ through the periodontal membrane to the apex of the root,* also through the Haversian canals in the bone." Talbot stated, "When simple gingivitis becomes chronic, the inflammation extends to the surface of the periodontal membrane. Necrosis occurs at the surface. *In the deeper tissues that have become inflamed pus cells also are found.*" Fish says, "The irritant . . . . is carried down the lymphatic vessels. These run under the epithelium lining *the gingivæ through and down the periodontal membrane* on their way to the lymph glands. The cellular infiltration spreads along the

*From the Foundation for Dental Research of the Chicago College of Dental Surgery, Chicago.
[1]Read before the International Association for Dental Research, March, 1941, St. Louis, Missouri.

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course of these vessels. This condition of chronic lymphangitis is shown to be responsible for the sequence of events recognized clinically as 'chronic gingivitis'." Merritt claims that, "Chief among the clinical symptoms of periodontoclasia are inflammatory reactions in the gingivae and pericementum."

Another group endorses the same concept as the first with the reservation that, occasionally, the inflammation extends into the external surface of the alveolar process or into the bone marrow spaces [F. B. Noyes,[10] E. D. Coolidge[8]]. Coolidge states that infiltration spreads into the tissue along the periodontal fibers or, exceptionally, along the outer border of the alveolar process. Noyes distinguishes between infections of the gum which are associated with deposits of calculus, and those which are not. Cases of gingivitis associated with deposits of calculus show "destruction of the bone under the periosteum, but the tissue between bone and cementum remains normal in character and appearance and shows no indication of infection." In cases of gingivitis without deposits of calculus the infection follows "the lymphatic capillaries from the connecting tissue papillae under the epithelium lining the gingival space which proceed toward the apex, passing through the fibers of the ligamentum circularis; and, between the bone and the cementum in the peridental membrane, there is inflammatory reaction. There is destruction of bone on the peridental membrane side of the alveolar process and formation of new bone on the periosteal side."

A third group of authors assumes that, while in the majority of cases of gingivitis the periodontal membrane is free of inflammatory reaction, in advanced cases the periodontal membrane may be affected. To this group belong K. H. Thoma and H. M. Goldman,[14] W. W. James and A. Counsell,[6] H. K. Box,[2] and R. Kronfeld.[7] Thoma and Goldman state, "In the marginal type of parodontal disease the peridental membrane is not affected except by a gradual progressive disintegration as pocket formation increases and bone is resorbed." James and Counsell state that the periodontal membrane remains free of inflammatory cells except in advanced cases, extension occurring from the bone marrow. "The path of invasion passes through the gum-corium to the alveolar bone, involves the bone marrow and later, the periodontal membrane." They further state that, "A study of the extension of cellular infiltration provides little evidence in support of the view that lymphatic vessels, from the corium, pass into the periodontal membrane." Box confirms the findings of James and Counsell and states that, "An extension of the cellular infiltration into the medullary spaces of the supporting bone is constant. It is noteworthy, on the other hand, that in the periodontal membrane these marked cellular reactions are, as a rule, absent or slight except in advanced conditions."

Kronfeld summarizes his observations on the problem in question as follows: "Any gingivitis that involves more than the superficial gingival tissues is likely to cause resorption of the bone at the alveolar crest. The mechanism of this bone resorption is the same as in every inflammatory process occurring in the neighborhood of bone: the inflammation causes hyperemia of the soft tissues and resorption of the adjacent bone. There is no true osteitis or osteomyelitis." At another place he says, "Inflammatory bone resorption in gingivitis is confined to the alveolar crest; the deeper bone and periodontal membrane are intact."

The last group is difficult to classify. Their statements are vague or controversial. In this group belong P. R. Stillman and J. O. McCall.[12] They say, "Periodontitis is seen to be characterized in its pathology by an inflammation beginning in the marginal gingiva." And at another place, "Alveolar resorption occurs almost entirely at the crest, and does not involve the inner wall of the socket until the disease has progressed well toward the tooth apex."

MATERIAL

The following table indicates the number, age and protocol number of the jaws in the collection of the Foundation for Dental Re-

Figure 1—Distal surface of the mandibular left second bicuspid of a 64 year old person. Inflammation (c) is progressing along the periosteal surface of the alveolar bone. Resorption of bone is occurring. The periodontal membrane (f) is free of inflammation. (a)—Epithelial proliferation. (b)—Ulceration of epithelium. (d)—Remnant of bony spicule between two marrow spaces containing edematous connective tissue. (e)—Alveolar bone proper.

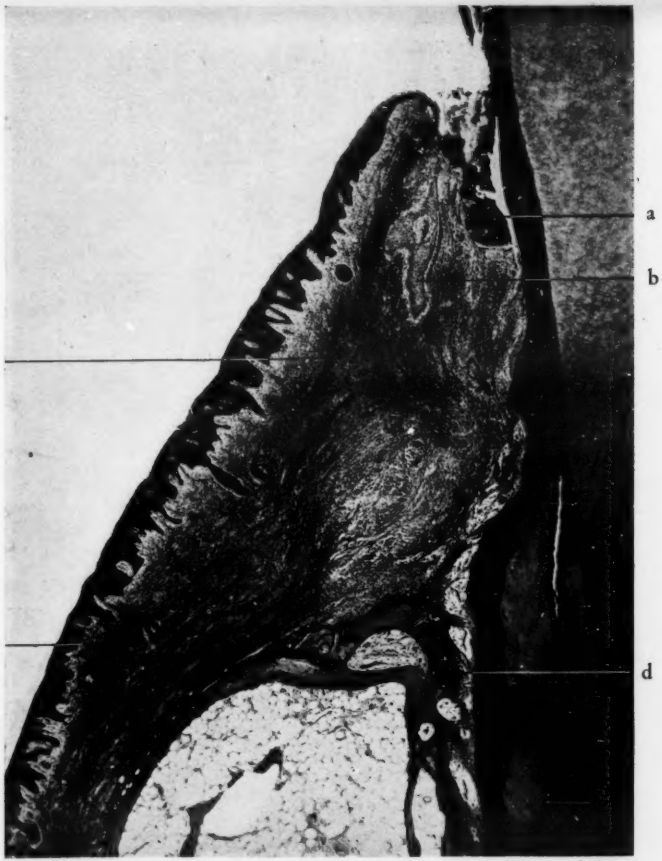

Figure 2—Buccal side of a maxillary left third molar of a 64 year old person. (a)—Deep pocket with calculus deposition. (b)—Epithelial projection into inflamed connective tissue. The inflammation is progressing along the outer surface of the alveolar crest and not into the periodontal membrane (d). The alveolar crest is resorbed from the periodontal surface. (c)—Accumulation of inflammatory cells in the subepithelial tissue.

search of the Chicago College of Dental Surgery which were used for this report:

No. of Jaws	Age	Protocol No.	No. of Jaws	Age	Protocol No.
1	11	62	2	51	34, 52
1	16	19	3	52	45, 59, 74
1	24	54	3	54	28, 35, K28
1	29	60	1	55	33
1	32	49	1	56	81
1	36	44	1	60	58
2	38	43, 38	2	63	43, 82
1	39	53	1	64	30
1	42	41	1	67	38
1	45	32	1	74	67
1	48	66	1	45-55?	5
3	50	50, 20, 70			

This investigation includes sections most of which were made through the center of the teeth in both buccolingual and mesiodistal directions. Cases were selected to illustrate characteristic types, degrees of inflammation of the gingiva, and ways in which the inflammation progresses into the supporting structures.

HISTOLOGICAL FINDINGS

Fig. 1 shows the distal surface of the mandibular left second bicuspid of a 64 year old person who died of uremia. This tooth is the most posterior of the remaining teeth on this side of the mandible since all molars have been extracted.

The depth of the pocket in the region illustrated is almost zero. The epithelial covering of the gingiva shows a projection of epithelium at *(a)* and ulceration at *(b)* close to the surface of the tooth. The chronic inflammation from the ulcerated area is progressing along the periosteal surface of the alveolar bone. The amount of bone resorption which occurred last may be calculated from the remaining bone spicule *(e)* bordering the periodontal membrane *(f)*. At *(d)* a bone spicule which separated two marrow spaces has escaped resorption. On both sides of the spicule there is edematous connective tissue resembling that found in the superficial bone marrow spaces in cases of inflammation of the gingiva. The gingival surface of the bone is bordered by resorption lacunae containing osteoclasts with one or many nuclei. The periodontal membrane is free of inflammation. The apposition of bone and cementum is progressing normally.

Figure 3—Lingual side of the lower right cuspid of a 54 year old person. Inflammation (c) is progressing toward the periosteal surface of the alveolar crest which is partly resorbed. Blood vessel has penetrated from the bone into the periodontal membrane. Accumulation of a few cells of lymphocytic type around the blood vessels (a); otherwise, the periodontal membrane is free of inflammatory cells. (b)—Alveolar bone proper which has escaped resorption.

Fig. 2 was obtained from the buccal side of the maxillary left third molar of the same 64 year old person from whom the previous specimen was taken. The left third molar on this side had been without its antagonist for a long time since the mandibular molars apparently were extracted a considerable period before the death of the individual.

At *(a)* a fairly deep pocket is present showing calculus deposited on the tooth surface in the pocket. At *(b)* a projection of epithelial cells extending into the underlying tissue can be seen. Adjacent to this area the epithelial covering of the pocket is ulcerated. The infiltration of chronic inflammatory cells is directed against the outer surface of the alveolar bone. At *(c)* a denser accumulation of inflammatory cells can be observed. A great part of the alveolus is already resorbed. That this resorption is still progressing is indicated by a resorption line which borders the gingival surface of the alveolar crest. The marrow in the adjacent marrow spaces is formed by edematous connective tissue. Chronic inflammatory cells are scattered around the enlarged blood vessels and bone is being deposited along the inner wall of the marrow spaces. Otherwise, the bone marrow space, which is extremely wide, is filled with fat marrow. The osteoporosis of the alveolus might be due to the long period the tooth has been in disuse.

In the periodontal membrane no inflammatory reaction can be observed. Apposition of bone is progressing on the periodontal face of the alveolar wall.

Fig. 3 shows the lingual side of the mandibular right cuspid of a 54 year old person. The chronic inflammation is progressing toward the periosteal surface of the alveolar crest. The chronic cellular infiltration is not in direct contact with the surface of the bone. A thin layer of connective tissue separates them. A part of the alveolar crest is resorbed. The resorption is progressing in some areas and in some places is reversed by apposition of bone. The details concerning these conditions differ in the various sections of the series. In some sections the surface of the bone is formed by a resorption line, whereas, in other sections an uncalcified layer of bone of varying thickness covers the resorption line. The amount of bone resorbed is equal, at least, to the height of the alveolar bone bordering the periodontal membrane which has escaped resorption so far.

A blood vessel at *(a)* has penetrated from the bone into the periodontal membrane. In the loose connective tissue around the blood vessel and between the bundles of the principal fibers a few round cells have accumulated. This slight cellular gathering follows the blood vessels from the bone marrow into the periodontal membrane and cannot be traced directly from the gingival margin. Otherwise, the periodontal membrane is free of inflammatory reaction.

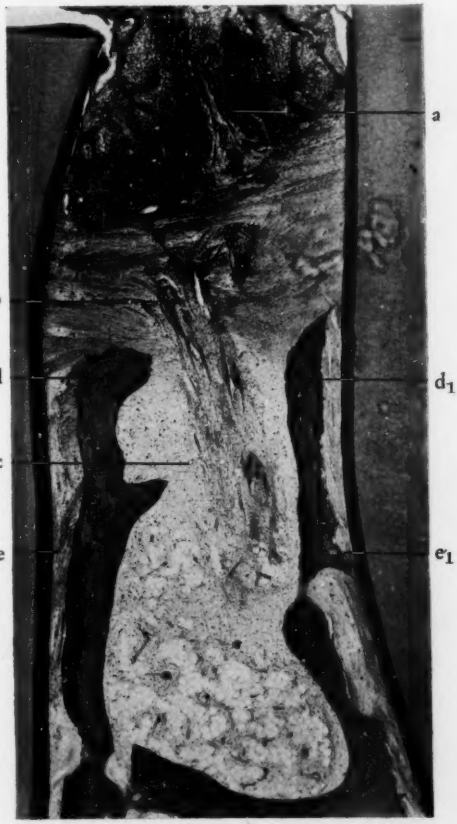

Figure 4—Space between first and second mandibular molars of a 16 year old boy. There is severe chronic inflammation of the papilla. The inflammation (a) follows the course of the blood vessel (b) into the bone marrow (c). The periodontal membrane (e,e_1) in this region is free of inflammatory reaction. The bone marrow shows edematous and fibrous changes as a reaction to the inflammation. (d,d_1)—Alveolar crest. (e_1) Area of pressure due to tipping of the second molar.

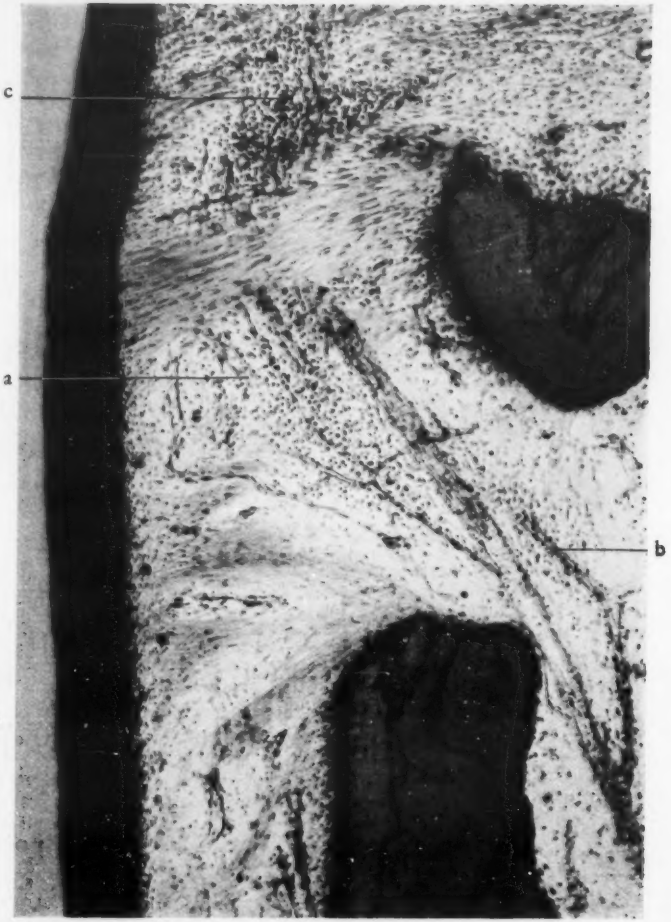

Figure 5—Higher magnification of alveolar crest from the same specimen as Figure 4 but from another section of the series. Slight inflammation (a) in the periodontal membrane around a blood vessel (b) passing from the bone marrow space into the periodontal membrane. This area of infiltration is in connection with the inflammation at the orifice (c) of the periodontal membrane.

Figure 6—Distal side of a maxillary right second bicuspid of a 54 year old person. "Parodontal abscess" (a). The inflammation follows the course of the blood vessels (b) into the bone. Resorption of the alveolar crest. The periodontal membrane (c) is free of inflammation. (Courtesy of B. Gottlieb, B. Orban, M. Diamond 5.)

The following two photomicrographs were obtained from the interdental spaces of the right mandibular molars of a 16 year old boy who died of cysto-pyelonephritis and prostatic abscess.

The distal half of the crown of the first molar has been destroyed by caries. The antagonistic upper first and second molars are missing. The first lower molar has no antagonist and only the most distal part of the crown of the second lower molar occludes with the upper third molar. The loss of the contact point by decay has caused a tipping of the second molar mesially, resulting in a traumatic pressure on the mesial side of the periodontal membrane of the mesial root. The pathology resulting from this occurrence is visible in the following photomicrograph, Fig. 4. In area *(e_1)* the pressure has caused a hyalinization of the periodontal membrane while the bone apically and occlusally to this area shows resorption on the side of the periodontal membrane.

The severe inflammation of the gingivae is probably due to the irritation resulting from the decay of the first molar. While the inflammatory cells infiltrating the epithelium are mainly leucocytes, the cells which have accumulated along the vessels are of the lymphocytic type. The course of the vessels can be traced from the subepithelial connective tissue, through the bundles of the transseptal fibers which stretch between the two teeth, into the bone marrow space. The bone marrow shows typical reactive changes, which decrease farther from the center of infection. In the marginal region the bone marrow consists of fibrous connective tissue. The fibers are separated by edema. We see inflammatory cells scattered through the fibrous connective tissue and accumulations of them along the blood vessels. In the gingival part of the bone marrow only a few fat cells remain; in the apical part continuous areas are formed by fat cells. These areas are separated by strips of fibrous connective tissue. The periodontal membrane is free of inflammatory cells.

A slightly different picture can be observed in Fig. 5 which is taken from another section of the same series. The bone trabecula which forms the gingival border of the alveolar crest shows resorption on the surface. Apically to this trabecula a blood vessel *(b)* passes from the bone marrow into the periodontal membrane. Along the delicate branches of this vessel we see a chronic infiltration *(a)*. This infiltration is connected with a group of inflammatory cells at the orifice of the periodontal space *(c)*. Both areas of infiltration are separated in this section by apparently normal fiber bundles. Here also the inflammatory reaction follows the blood vessels which run a short distance through the periodontal membrane. Otherwise, the periodontal membrane is free of infiltration.

Fig. 6 shows the distal side of a maxillary right second bicuspid of a fifty-four year old person.

Half-way between the oral mucosa and the alveolar crest an abscess (retention cyst) has developed. In other sections of this same specimen it can be observed that this abscess is in communication with a deep pocket. We can differentiate three layers in this so-called lateral abscess: a center zone consisting mostly of leucocytes, a lighter stained layer of epithelial cells lining the cavity and an outer layer of chronic granulation tissue. From the outer layer of the abscess a stream of inflammatory cells runs in the direction of the alveolar crest following the course of the blood vessels. The alveolar bone is being resorbed and the chronic inflammatory reaction passes into the bone marrow. In the adjacent bone marrow spaces a fibrous marrow with scattered chronic inflammatory cells has replaced the fatty marrow which still remains in the areas most distant from the inflammation. The periodontal membrane is free of inflammatory cells.

Fig. 7 shows the interdental space between the right mandibular second bicuspid and first molar of a 50 year old person who died of bronchial pneumonia. On the distal side of the second bicuspid a pocket 4 mm. deep *(a)* (measured without taking into consideration the amount of shrinkage due to fixation) has developed. The condition of the supporting tooth structures is similar to that found in gingivitis without deep pocket formation. The gingival alveolar plate *(b)* is resorbed and the resorption is still in progress. The bone marrow of the gingival part of the alveolar process is formed by edematous fibrous tissue. The chronic inflammation has progressed into the bone marrow but the periodontal membrane *(c)* is free of inflammatory reaction.

Figure 5—Higher magnification of alveolar crest from the same specimen as Figure 4 but from another section of the series. Slight inflammation (a) in the periodontal membrane around a blood vessel (b) passing from the bone marrow space into the periodontal membrane. This area of infiltration is in connection with the inflammation at the surface of the gingival margin.

of a 54 year old person. "Parodontal abscess" (a). The inflammation follows the course of the blood vessels (b) into the bone. Resorption of the alveolar crest. The periodontal membrane is free of inflammation. (Courtesy of Dr. Coolidge, H. Orban, M. Diamond D.D.S.)

Figure 7—Interdental space between the right mandibular second bicuspid and first molar of a 50 year old person. (a)—Deep pocket. Resorption of the gingival alveolar plate (b). Transformation of the fatty marrow into fibrous marrow. Inflammation progressing into bone marrow. Periodontal membrane (c) free of inflammatory cells.

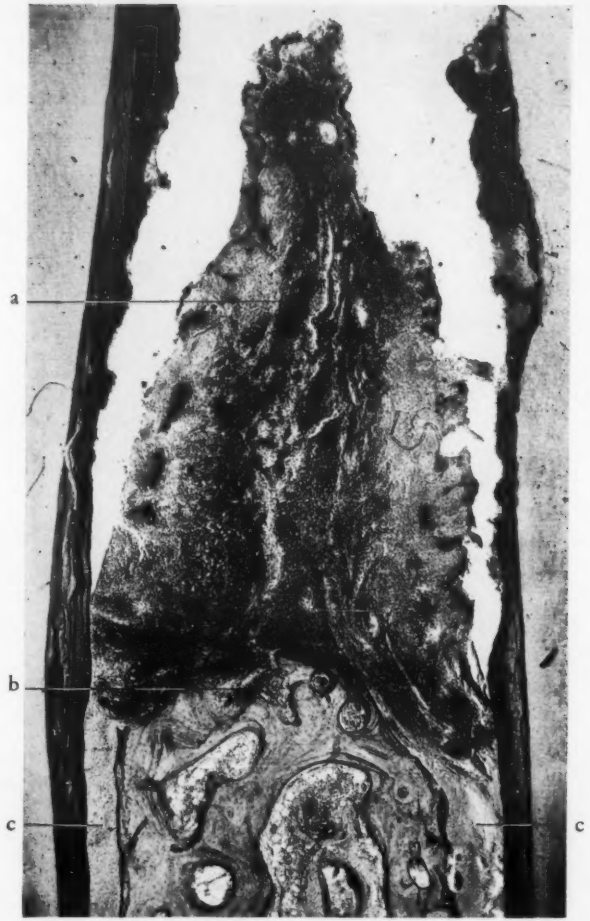

Figure 8—Interdental papilla (a) between the right maxillary lateral incisor and cuspid of a 40 year old man. Ulcerative gingivitis. Acute inflammation has penetrated (b) into the bone marrow spaces. The periodontal membrane (c) is free of inflammatory cells.

The previous specimens illustrated the condition found in cases of chronic inflammation of the gingiva. Figs. 8 and 9 show the findings in a case of acute gingivitis. The sections show the interdental space between the right maxillary lateral incisor and cuspid of a 50 year old man who died of heart failure.

Fig. 8 shows necrosis of the epithelium. Islands of epithelial cells are preserved in only a few small areas. Acute inflammatory cells *(a)* have penetrated the epithelium and subepithelial connective tissue and also prevail in the dense infiltration progressing to the alveolar ridge and deep into the bone marrow spaces. Pathologic conditions analogous to those characterizing an ulcerative gingivitis (Vincent's gingivitis) are present in the gingiva of all teeth in this case. While the bone marrow spaces show acute and chronic inflammatory infiltration, the periodontal membrane *(c)* is free of any inflammatory cells.

Fig. 9 shows the sharp demarcation of acute infiltration *(b)* toward the periodontal membrane *(c)* and the progressing resorption of the alveolar bone from the gingival side *(a)*. While the periodontal membrane *(c)* is free of inflammatory reaction, the bone marrow space *(d)* at the same level shows round cell infiltration.

Figure 8—Interdental papilla (a) between the lateral incisor and cuspid of a 40 year old man. Interstitial gingivitis. Acute inflammatory infiltration progressing into the bone marrow. The periodontal membrane (c) is free of inflammatory cells.

Figure 7—Interdental space between the right mandibular second bicuspid and first molar of a 50 year old person. (a) Deep pocket. Resorption of the gingival alveolar plate. (b) Transitional epithelium. (c) Acute inflammatory infiltration progressing into bone marrow. Periodontal membrane (c) free of inflammatory cells.

SUMMARY OF FINDINGS

Histological investigation of the effects of chronic and acute gingivitis on the supporting structures revealed the following facts:

1. Resorption of the alveolar crest on the periosteal side leads first to destruction of the supporting alveolar bone and later of the alveolar bone proper.

2. Inflammation is found following the course of the blood vessels into the bone marrow spaces.

3. The periodontal membrane, as a rule, is free of inflammatory reaction, regardless of the degree of inflammation. Slight inflammatory reactions in the periodontal membrane can be observed only in exceptional instances. In the few cases found the inflammation followed the blood vessels from the bone marrow into the periodontal membrane and was independent of the severity of the inflammation in the gingiva.

4. The fatty marrow of the alveolar crest was replaced by edematous, fibrous marrow.

DISCUSSION

This investigation revealed that the distribution of the inflammatory reaction follows the course of the blood vessels. This observation needs elaboration in two respects:

1. What is the distribution of the blood vessels of the dental apparatus, and

2. Why are the inflammatory cells found predominantly in the neighborhood of the blood vessels?

The distribution of the blood vessels can be studied best in specimens where the vessels have been injected with special stains. This method was used to demonstrate the blood and lymph vessels of the gingiva by Schweitzer[11] and also by Noyes.[10] In this investigation the distribution of blood vessels was studied in sections where stasis had distended the lumen making it easy to follow their course. An instructive section of this kind is shown in Fig. 10 which shows the left central and lateral mandibular incisors. The course of the vessel *(a)* can be followed almost the entire length of the septum. We see at *(b)* the ramus which emerges in the subgingival tissue, and at *(c)* branches which penetrate into the periodontal membrane. Other sections of the series show the ramification of the interalveolar vessels which supply the periodontal membrane in other areas of the interdental septum. From such specimens we can develop a concept of the vascular supply of the tooth and its supporting structures. Our findings confirmed in all respects the description given by E. Zuckerkandl[15] in 1891.

The main branch supplying the tooth and the tooth supporting structures divides into two groups: the dental arteries and the interalveolar arteries. The dental arteries are very delicate vessels. Just before entering the apical foramen into the pulp they give off small branches which supply the immediate neighborhood of the foramen. The interalveolar arteries are larger vessels, their diameter being many times greater than that of the dental arteries. The interalveolar arteries passing through the interdental septa to the alveolar crest divide into small branches. These branches are called alveolar perforating branches. They reach the periodontal membrane through many openings in the lateral wall of the alveolus. The interalveolar arteries penetrating the alveolar crest through gaps in the bone extend into the gingivae (gingival perforating

Figure 9—Higher magnification of area between (b) and (c) in Figure 8. Resorption of the alveolar crest (a). Progress of acute inflammatory cells (b) is limited by principal fibers of the periodontal membrane (c), which is free of inflammatory cells. (d)—Inflammatory cells in the bone marrow.

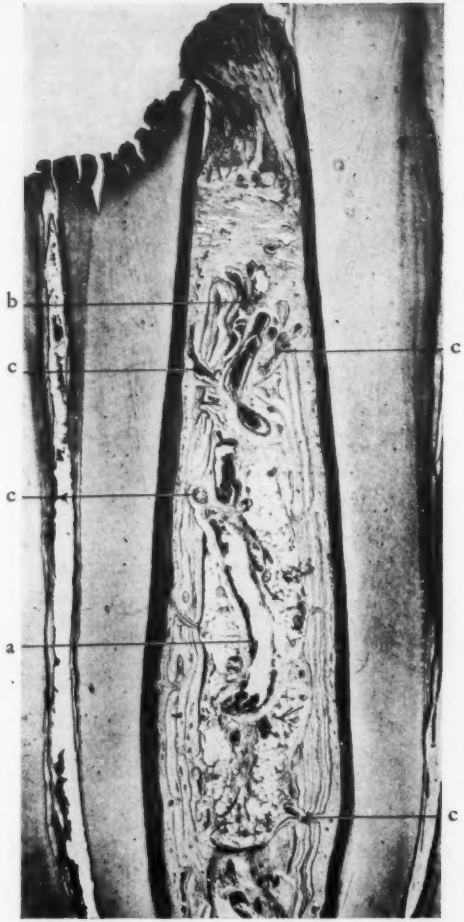

Figure 10—Interdental septum between central and lateral mandibular incisor of a 64 year old person. (a)—Interalveolar artery running in the center of the septum giving off branches to the periodontal membrane and gingival margins. (b)—Gingival penetrating arteries. (c)—Alveolar penetrating

branches) where they divide into fine branches and capillaries. The capillaries can be followed into the connective tissue papillae of the mucous membrane of the gingivae.

Beside the branches of the interalveolar arteries the lingual and buccal gingiva is supplied by means of independent vessels which lie in the connective tissue between the epithelium and the periosteum of the mandibular and maxillary bone. We may assume that there are anastomoses between the vascular networks of the gingiva and the periodontal membrane, and between the various regions of the periodontal membrane nourished by alveolar perforating arteries.

From the present study it was found that the inflammatory reaction follows the distribution of the blood vessels.

Zuckerkandl's description and our own studies concerning the distribution of the blood vessels revealed that a connection between the gingival and periodontal area is of only minor importance. These combined facts explain the observation that the periodontal membrane is generally free of inflammatory reaction in cases of gingivitis, the reason being that the inflammation follows the course of the blood vessels.

The other item which needs elaboration is the question why inflammatory cells are found predominantly in the neighborhood of the blood vessels.

It has been shown by means of injection of different dyes (H. Loeschke,[8] G. Schweitzer,[11] F. B. Noyes[10]) that each capillary is surrounded by a lymph sheath or network of lymph vessels by means of which material may be absorbed. These lymph vessels are the physiologic roads for the transportation of substances from the tissues. Under pathologic conditions they are the routes through which bacteria, toxins or tumor cells are carried to different parts of the body.

These vessels are also surrounded by loose connective tissue. The loose connective tissue contains cells possessing the potentiality of being transformed into defensive elements.

Accepting the foregoing interpretation of the function of the lymph vessels and the loose connective tissue, we can understand why the inflammatory reaction is observed along the course of the blood vessels. Toxins produced by the bacteria are absorbed and penetrate into the lymph vessels. Undifferentiated connective tissue cells transform into inflammatory cells due to toxic irritation. At the same time the fatty marrow is transformed into fibrous marrow by the same factors.

The pathological changes brought about by irritation of the gingivae may be termed gingivitis as long as they are confined to the soft tissues. If the changes lead to resorption of the alveolar crest, an inflammatory reaction following along the blood vessels into the bone marrow spaces, and a transformation of the fatty marrow into fibrous marrow, the pathological picture should be classified as osteitis.

SUMMARY

The progress of chronic and acute inflammation of the gingiva into the supporting structures was studied histologically in jaws ranging from 11 to 74 years which were secured at autopsy.

A review of the literature revealed different opinions in regard to how inflammation of the gingivae extends into the supporting structures.

The histological studies revealed the following findings: Chronic inflammation of the gingiva progresses, following the course of the blood vessels, into the bone marrow spaces and on the periosteal side of the alveolar bone; only in exceptional instances does it penetrate into the periodontal membrane. Resorption of the alveolar crest from the gingival side leads to destruction, first, of the supporting alveolar bone and then the lamina dura. The fatty marrow is replaced by fibrous marrow.

The distribution of the blood vessels explains the pathological picture. The effect of gingivitis on the supporting structures is interpreted as osteitis.

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Resorption of the alveolar crest (a). Progress of acute inflammatory cells (b) is limited by principal fibers of the periodontal membrane (c), which is free of inflammatory cells. (d)—Inflammatory cells in the bone marrow.

Figure 10—Interdental septum between central and lateral mandibular incisor of a 64 year old person. Inflammation can be seen in the center of the septum giving off branches to the periodontal membrane and gingival margins. (b)—Gingival penetrating arteries. (c)—Alveolar perforating

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SUPPURATIVE INFLAMMATION OF THE GUMS, AND ABSORPTION OF THE GUMS AND ALVEOLA PROCESS*

By JOHN W. RIGGS, M.D., F.A.A., D.S., *Hartford, Conn.*

(Copied from *The Pennsylvania Journal of Dental Science*, **3**:99, 1876)

This disease is called by many the disease of old age, as formerly it was more particularly noticed in persons of advanced years, but at the present day we find the middle aged, and even the young, affected by it. Many of the textbooks of our specialty consider it hereditary, or constitutional, or bone disease, arising from a scrofulous diathesis. The result, from whatever cause, is most disasterous to the teeth, and in many cases to the health of the patient, for one by one the teeth become loose from loss of bony support and are plucked out as an intolerable annoyance. If the inflammatory action be great and involve most or all the gum embracing the teeth, pus tinged with blood exudes from around the necks of the teeth on the slightest pressure of the lips or tongue, or in mastication. The oral secretions become vitiated. present a viscid or sanious character, very abundant in quantity during the day, but much more so in the recumbent position of sleep. If the patient reposes on his side these exudations flow out of the corner of the mouth over the pillow and present in the morning a dried, yellow discoloration, often tinged with blood and covering a space as large as one's hand. If the patient reclines on his back the diseased mass flows back into the fauces and is unconsciously swallowed, there to work a greater mischief by breaking down the tone of the stomach and poisoning its contents. I apprehend there can be little question or dispute among physicians or physiologists as to the deleterious effect of this putrefactive ferment on the digestive organs. . . .

Notwithstanding this single opinion, the diagnosis of a number of cases for treatment fully sustains us in the former conclusions, confirmed, as they are, in every instance by the rapid recovery of the general health of the patients by restoring the particular health of the mouth.

None but the most vigorous constitutions can withstand this type of disease. The appetite begins to fail . . . A cup or two of strong coffee, and a few crackers, constitute the sole breakfast; languor and ennui are substituted for cheerfulness and vigor of mind and body; neuralgic pains sweep up through the face to both hemispheres of the brain, and are intensified by a recumbent position, thus banishing sleep . . .

The disease also aggravates, if it does not cause nasal catarrh. One of my patients, a physician, the best authority, of course, strenuously maintains that the restoration of his gums, teeth and mouth, to health, relieved him entirely of a virulent catarrh, upon which he had exhausted all known remedial agents.

One patient, besides great prostration from aggravated symptoms, became nearly blind. She could not see to read ordinary print, and could only tell the number of windows in a room by the mass of light from each. She could not walk up a flight of stairs without assistance. I had the pleasure, some three years ago, of presenting this patient for examination before that very enthusiastic society of workers, the Brooklyn Dental Association. She was then well, sufficiently so to walk several miles daily. Gums and mouth, healthy; appetite good, and fast improving in general condition. As this disease exists in a less annoying and less dangerous form for several years previous to the above aggravated symptoms, covering from ten to twenty years before it culminates in its miserable characteristic, the loss of the teeth or life, I have thought best to tabulate its progress by treating it under four heads or divisions, as follows:

First. Where the margin of the gums shows decided inflammatory action, with some absorption of its substance, and bleeding at the slightest touch of the brush.

Second stage. Where the inflammation extends down over the thinner alveolar border, causing absorption of the bone, as well as gum tissue, forming small pockets beneath the gum filled with pus.

Third. Where the diseased action takes deeper hold, involving the thicker portions of the process, absorbing it most rapidly nearest the tooth, causing the tooth to sway back and forth for lack of most of its bony support.

(Continued on page 105)

*Read before the American Academy of Dental Surgery, New York, Oct. 20, 1875.

To What Extent Should the Tissues Be Excised in Gingivectomy? *[1]

BALINT ORBAN, M. D., D. D. S., *Chicago, Illinois*

The question arises again and again—"How deep ought we to cut in gingivectomy and how much tissue should we remove." The aim of gingivectomy is to eliminate the deep gingival sulci and thus promote hygienic conditions. The gingival sulcus is a potential trap for the sediments of saliva, epithelial cells, leukocytes, mucin, and organisms of different types and kinds. These organic deposits form a matrix for calcium salts to be precipitated from the saliva, and thus calculus is formed. For this process there are always possibilities, even in the shallowest gingival sulcus, and this accumulation of foreign material in the gingival sulcus is the beginning of trouble. The mechanical irritation, as well as the formation of bacterial toxins, will produce an irritation in the sub-epithelial connective tissue, thus setting up an inflammatory reaction. This inflammatory reaction is present because of the irritation and it must be considered as a defensive action of the connective tissue against the invasion of foreign irritating substances, especially bacteria and their toxins. However, if the irritation becomes more and more extensive the inflammatory reactions will lead to undesirable tissue changes, such as hyperemia, local circulatory disturbances, development of acid tissue reaction, destruction of the surface epithelium and ulcer formation, also migration of leukocytes through the epithelium and ulcerated surface into the gingival sulcus, and thus a pus flow from the pocket. Due to the aggravated inflammation the epithelial attachment separates from the surface of the tooth at the bottom of the sulcus and the gingival sulcus becomes deeper; thus conditions gradually become increasingly worse.

In eliminating the pocket by surgery the gingival tissue outside the bottom of the pocket—that is the free gingiva—must be cut away. Fig. 1 shows an ulcerated papilla. The sulci on both sides are filled with tartar to the bottom of the pocket. The epithelium covering the papilla is partly destroyed and has partly proliferated into the underlying granulation tissue. This granulation tissue constitutes the defensive reaction of the connection tissue and it is found in various degrees everywhere in the gingiva where toxins penetrate.

In considering the case shown in Fig. 1, as a practical case, we would have to perform a gingivectomy from the bottom of one sulcus to the bottom of the sulcus on the other side—from (a) to (b).

Different operators follow different methods in performing gingivectomy. Some of them just cut away the gingiva outside the bottom of the pocket, and after stopping the bleeding, place a surgical pack over the cut area. Others scrape or curette all the tissues between the bottom of the pocket and the alveolar crest. There are still others who scrape, trim or chisel the bony margin. These different methods will be discussed at this time from the histopathological point of view. According to histological and clinical experience the first method—cutting the gingiva to the bottom of the gingival sulcus—with the necessary post-operative treatment, is the best method. Analyzing the conditions shown in Fig. 1, this method wins strong support. If the tissues are removed in a straight line, from (a) to (b), a thin layer of the granulation tissue remains on the surface. Below this thin layer a strong group of transseptal fibers are expanded between the two teeth and above the alveolar crest. In this case there certainly is no reason to scrape out the tissues lying between the bottom of the sulcus and the alveolar crest, and certainly no reason to touch the bone.

The granulation tissue exposed to the surface becomes reduced or eliminated by the post-operative treatment during the healing process of the wound. The development of granulation tissue is due to an irritation from the gingival sulcus and after the pocket is eliminated it will become de-

*Read at the Fifty-eighth Annual Meeting of the Maryland State Dental Association, Baltimore, May 5th, 1941.

[1]From the Foundation for Dental Research of the Chicago College of Dental Surgery.

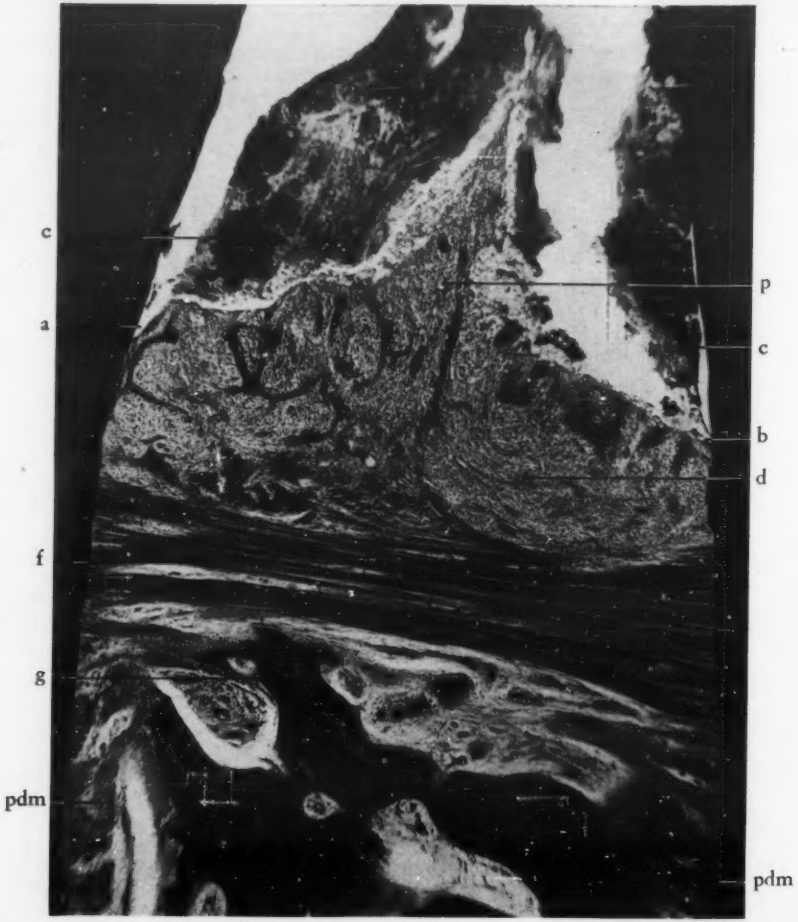

Figure 1—Interdental papilla (p). The bottom of the gingival sulcus (a-b) on the cementum. Calculus (c) fills the sulci. The epithelium of the papilla is very thin and missing in several areas. Below the epithelium granulation tissue (d) developed. Strong bundles of transseptal fibers (f) crossing over the alveolar bone (g). Periodontal membrane (pdm) free of inflammation.

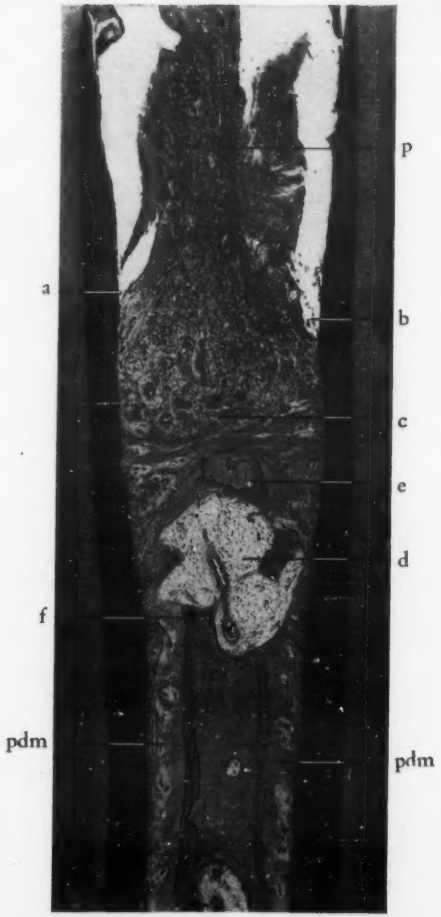

Figure 2—Interdental papilla (p). The bottom of the gingival sulcus (a - b) on the cementum. The inflammatory granulation tissue extends to the trans-septal fibers (c). At (d) the bone is resorbed and replaced by a loose connective tissue. A bone spicule (e) is lying free below the trans-

Figure 3—Deep gingival pocket (a) around a lower incisor. The gingival sulci on the adjacent teeth (b) are considerably shallower. The interdental spaces are filled with heavy calculus deposits.

creased in the process of wound healing. There is of course no objection to slightly scraping the surface of the exposed tissue in order to eliminate large masses of chronic inflammatory cells, which are composed mostly of plasma cells. The granulation tissue can be recognized by its redness, bleeding and softness, while fiber bundles will give considerable resistance. If large masses of granulation tissue are present they are superfluous after gingivectomy and we will encounter a faster healing tendency if they are eliminated. This scraping should be performed carefully as we do not wish to injure the connective tissue fibers below the granulation tissue. If the granulation tissue is not removed it will start to grow over the surface as a velvety dark red tissue granulation. It should be treated with astringent drugs such as zinc chloride (8%), trichloracetic acid (20%) or silver nitrate (concentrated in crystals) to prevent its overgrowth and to keep the epithelium from growing too rapidly over the still granulating wound surface.[1] This epithelium would be weak and thin and would not form a desirable surface covering. After the granulation has stopped the epithelium will form a strong continuous layer above the exposed connective tissue. It is much better

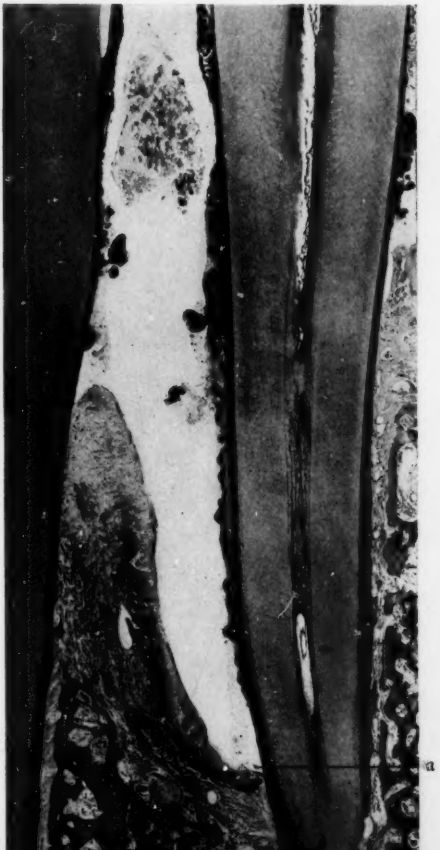

Figure 4—Deep gingival pocket (a) on the distal side of a lower incisor. The sulcus on the mesial side of the cuspid (b) and on the mesial of the incisor is in a much more favorable position, and shallow.

[1]See "Indications, Technique, and Post-operative Management of Gingivectomy," p. 89.

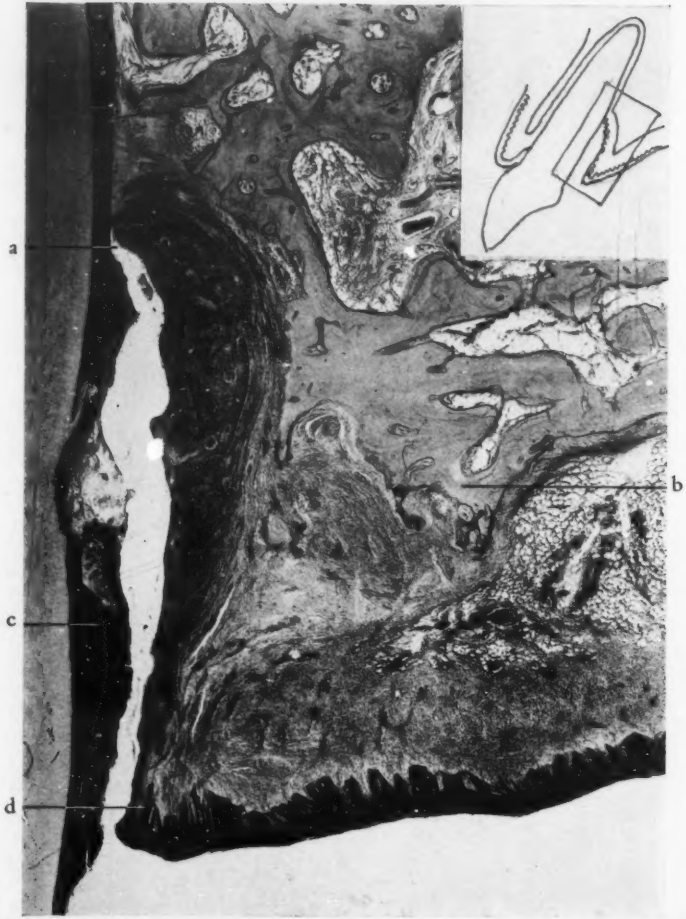

Figure 5—Deep gingival pocket on the lingual side of an upper incisor. The bottom of the sulcus (a) is above the crest of the bone (b). This bone is part of the hard palate and not of the alveolar bone. c: calculus in the sulcus. d: free gingival margin.

Figure 6—Deep gingival pocket on the lingual of a lower bicuspid. The bottom of the sulcus (a) is below the level of the bone margin (b). The bone margin is jaw bone and not alveolar bone. c: calculus in the sulcus. d: free margin of the gingiva.

to treat this granulating surface a few times (every second day for ten to fourteen days) than to scrape more than desirable, thus preventing regeneration of connective tissue fibers and the alveolar crest. Even if all the granulating tissue has been removed there will be some post-operative granulation which must be prevented from overgrowth if a satisfactory result is to follow the gingivectomy. No bone regeneration can be expected after radical removal of all tissues between bone and gingival sulcus.

Sometimes it is claimed that the alveolar crest is diseased in the case of gum inflammation (pyorrhea) and that the bone is infected—even necrotic. It was repeatedly stated by men trained in pathology, that in this disease the bone is not infected and never necrotic. The inflammatory reaction might penetrate into the marrow spaces of the bone, but not into the periodontal membrane. This has been clearly demonstrated by Weinmann (see p. 77). The penetration of the inflammation by no means indicates an infection in these areas. It only means that the toxins from the gingival sulcus have been transferred along the blood vessels—most probably in the lymph spaces —and thus into the deeper structures. The bone itself is not infected and not necrotic. There is no justification whatsoever for exposing this bone and operating on it in any way. It must be clearly understood that bone never is exposed in pyorrhea. The alveolar bone is always separated from the gingival pocket by connective tissue. If by exploring the pocket one feels the surface of rough naked bone, one can immediately make the diagnosis of osteomyelitis, and not of pyorrhea. Of course even in these cases the bone should not be operated on. It has also been claimed by men who advocate radical surgery that the edges of the alveolar crests are irregular and that sharp spicules will extend into the tissues and prevent healing. There is no justification for this claim. In Fig. 1, of Dr. Weinmann's report (p. 72), a sharp bone spicule forms the alveolar crest *(e)* while the rest of the bone was resorbed by the extension of the inflammatory reaction outside of the bony crest. However, in treatment of gingival disease, if we eliminate the irritation we can expect the inflammation to diminish and the bone to regenerate up to the end of the bony spicule. If this bone had been removed during the operation no regeneration of bone could be expected and some of the healthy attachment would have been destroyed. The same thing is true in the specimen shown in Fig. 2. The bottom of the gingival pocket is at *(a)*, and *(b)*. The gingivectomy should be performed along a line connecting these two points. If tissue is curetted after operation this should not go beyond the point *(c)*, where strong connective tissue bundles are found in good functional order. Alveolar bone has been resorbed at *(d)*, but a bony island *(e)*, was left to form the uppermost crest of the bone. If in operating, the connective tissue bundles at *(c)*, and the bone island at *(e)*, are removed, the alveolar crest is lowered to *(f)*, and no regeneration can be expected. If operated cautiously one can expect bone regeneration at the site of the loose connective tissue between *(e)*, and *(f)*.

The condition is similar in the case shown in Fig. 4 of Dr. Weinmann's report (p. 75). Due to the gingival inflammation a part of the alveolar crest has been resorbed but the alveolar plates remained on both sides *(c)*, and *(d)*. Between these two plates the bone has been resorbed, probably due to penetration of the inflammatory reaction into the bone marrow *(c)*. If in operating we should remove the two bone plates we shorten the alveolus and thus damage the supporting mechanism. If the irritation is removed and good hygienic conditions rendered, regeneration of bone and thus a strengthening of the supporting structures can be expected. The same consideration must lead us in all our cases. (See also Figs. 3, 5, and 8—Dr. Weinmann, pp. 74, 76, 78).

Somewhat different in appearance, but similar in consideration, is the case shown in Fig. 9 of Dr. Weinmann's report (p. 80). An acute ulcerative inflammation attacked the entire interdental papilla *(a)*. The epithelium is entirely destroyed; the surface of the papilla is almost entirely necrotic and the inflammation penetrates to the surface of the bone *(b)*, and into the marrow spaces. There is no healthy connective tissue between bone and granulation tissue. Truly, there is no granulation present and the inflammation is largely acute. This type of case must be treated conservatively by elim-

Figure 5—Deep gingival pocket on the lingual side of an upper incisor. The bottom of the sulcus (a) is above the crest of the bone (b). This bone is part of the hard palate and not of the alveolar bone. c: calculus in the sulcus. d: free gingival margin.

Figure 6—Deep gingival pocket on the lingual of a lower bicuspid. The bottom of the sulcus (a) is level with the level of the bone margin (b). The bone margin is part of the lower bone. c: calculus in the sulcus. d: free margin of the gingiva.

inating infection and irritation, also by the use of suitable drugs and probably systemic treatment. The acuteness of the inflammation must be reduced. In these extreme cases we also can expect tissue regeneration if we do not use radical methods in treatment.

The only time we might be compelled to operate on bone is in cases where the bottom of the gingival crevice is below the crest of the bone. This might be the case between two teeth, as shown in Fig. 3. Here the pocket on one tooth is close to the apex of the tooth *(a)*, while on the adjacent tooth the pocket is considerably shallow *(b)*. Fig. 4, shows a similar condition—there is a deep pocket on the distal side of a lower lateral incisor *(a)*, while on the mesial side of the same tooth and on the mesial side of the cuspid *(b)*, the pocket is shallow. If in these cases we want to eliminate the pocket, we must not only cut the gingiva but also the bone. It would be necessary to cut so much bone away that we would render the bony crest horizontal (along the line a). However, as Gottlieb has claimed for years,[1] by doing this we certainly afflict damage to the tooth which has the shallow pocket and a fairly good supporting mechanism, in favor of a tooth which has a questionable future. The teeth, shown in Figs. 3 and 4, might better be extracted than treated in this radical way. If for any reason it seems necessary to postpone the extraction, the gingivectomy could be carried out, according to the rules formerly described, from the bottom of one pocket to the bottom of the other pocket—not eliminating it entirely, but widening it and thus making it possible to maintain a hygienic condition. This of course is not always ideal but it is better than afflicting damage to a relatively healthy structure in favor of a diseased one, without a definitely favorable prognosis. Quite often such teeth can be kept in good condition for many years.

The bottom of the gingival sulcus might be above the bony crest in the region of the lingual side of the upper teeth where the hard palate is in the back of the alveolar bone. Fig. 5, shows one of these cases in the region of an upper incisor. To eliminate this pocket it is necessary to remove part of the bone. However, we can do this without any hesitation as that bony crest does not serve the purpose of supporting the tooth. This is also true in the region of the lower molars on the buccal, or the bicuspids on the lingual side, in the presence of exostosis on the lingual side of the mandible. Fig. 6, shows a microscopic picture of a lower bicuspid where the bottom of the pocket is below the bony crest, but this crest is not alveolar bone—it is a part of the jaw bone and does not support any teeth. To eliminate this pocket the crest of the bone must be leveled off in a line below the bottom of the gingival crevice.

Figure 7—Roentgenogram showing exposed bifurcation of a lower molar. The space has been enlarged through gum resection and trimming of the bony septum.

There is another instance in which the bone should be removed and that is in the lower molars where the bifurcation is involved. These exposed bifurcations are difficult to keep clean and they often lead to acute abscess formations. In cases where the retention of a tooth with an exposed bifurcation is indicated, we can widen the space in the bifurcation by cutting away a part of the inter-radicular bone septum. Fig. 7, shows a roentgenogram of a case of this type. The patient is now able to keep the wide bifurcation clean by using a pipe cleaner twice daily.

By no means should it be suggested that gingivectomy is the only means to eliminate a periodontal pocket. Curettage, polishing of the teeth, and proper home care often make gingivectomy unnecessary. The aim of this short paper is to define a limitation as to how far surgery should be carried and to support the statements by histopathological evidence.

180 N. Michigan Avenue

[1]Gottlieb, B.: Z. *Stomat.* 22:1. 1924.

Indications, Technique, and Post-Operative Management of Gingivectomy in the Treatment of the Periodontal Pocket*[1]

By BALINT ORBAN, M. D., D. D. S., *Chicago, Illinois*

In the treatment of periodontal pockets the primary aim is to eliminate them. To fully appreciate this apodictical statement we must understand the nature and development of these pockets. The normal gingival pocket or sulcus is a V shaped space which develops, after tooth eruption, between the free margin of the gingiva and the tooth surface. This physiological sulcus is very shallow and the gingivae is without any appreciable inflammation. The bottom of this gingival sulcus is on the tooth surface, where the epithelium of the mucous membrane joins the enamel epithelium which is attached to the surface of the enamel, Fig. 1.

The biological fact that the bottom of the gingival sulcus is at the point where the epithelium is attached to the tooth makes this area a vulnerable spot. The epithelium at the bottom of the sulcus is constantly exposed to irritation and therefore a continuous degeneration of cells takes place at this spot. Due to the bacteria which are always present and the formation of calculus in the gingival sulcus, the gingival tissues become inflamed and this inflammation is the first step in the development of a pyorrhea pocket. The irritation produces hyperemia and edema of the gingivae and this swelling of the gingival tissues deepens the pocket. In this slightly deepened pocket more deposits form, bacteria lodge, and irritation progresses. As a result of this inflammatory reaction the epithelium which is attached to the tooth surface degenerates at the bottom of the pocket and is separated from the surface, leading to further deepening of the sulcus. In this way the bottom of the gingival sulcus shifts apically on the surface of the tooth, while the gingiva is swollen and hyperemic. Both of these conditions lead to deepening of the pocket. If gingival inflammation accompanies the presence of a deep pocket and the latter's contents are of a purulent nature, it is commonly called pyorrhea, Fig. 2.

It should be understood without any doubt that we do not consider the shifting of the bottom of the gingival sulcus to

Figure 1—Normal gingival sulcus. The bottom of the gingival sulcus (a) is on the enamel (b). The end of the epithelial attachment (c) has left the cemento-enamel junction (d) and migrated along the cementum. There is only a slight inflammation in the subepithelial connective tissue (e). The free margin of the gingiva (f) is covered by horrified epithelium. The enamel cuticle (g) is floating in the space formerly occupied by the enamel.

*Read at the Fifty-fifth Annual Meeting of the Colorado State Dental Society, Colorado Springs, June 20th, 1941.

[1]From the Foundation for Dental Research of the Chicago College of Dental Surgery.

be purely the result of an inflammatory reaction. The fact alone, that epithelium is attached to a calcified hard structure, explains the continuous separation of the epithelium from this surface due to degeneration of the epithelial cells at the bottom of the gingival sulcus. Biologically this one fact explains the continuous shifting of the bottom of the gingival sulcus along the surface of the tooth. This biologic process is only complicated by the presence of foreign material such as calculus, bacteria, shedding cells, food particles, etc., in the depth of this sulcus.

Figure 3—Gingival pocket explorer.

There are periodontal pockets which develop in a different way and these cases we consider to be due to some systemic disturbance—endocrine, nutritional, or of some other nature. In these cases the inflammation of the gingivae is most probably of a secondary nature following the formation of the deep pocket. These systemic cases are relatively rare and in their clinical consequences do not differ from the inflammatory type. The purpose of treatment of all deep pockets is the same—they must be eliminated.

Figure 2—Deep pocket (a) in the interdental space between two bicuspids. The continuity of the pocket epithelium (b) is broken at several spots exposing connective tissue and forming ulcers. The pocket on the outer side of the space is not quite as deep (c). The free margin of the gingivae (d) is covered by epithelium. At (e) the alveolar bone is seen.

If the pocket is not of extreme depth, that is less than about 3 mm., a conservative treatment would be indicated. This treatment consists of most careful scaling, polishing, subgingival currettage, and proper home care. This procedure will, in a large number of cases, lead to complete healing. The better the scaling technique and the better the home care—the larger the number of cases successfully treated.

Even in cases of deeper pockets a conservative treatment might be successful, but the time and effort needed to complete a case in this manner might be so extensive that the cost would be more than most patients could afford. Gingivectomy is the better choice in these cases. There is a wide range of possibilities in the selection of suitable cases. We can perform a gingivectomy in any and every case where a pocket has to be eliminated. In ten to fourteen days after the operation one can expect the case to be completed.

The best practical procedure is first to eliminate the acute condition of the gingival inflammation and thus reduce bleeding. This is accomplished best by scaling a day or two previous to the gingivectomy. This scaling need not be the most thorough, but all rough parts of deposits should be removed.

To perform the gingivectomy we should first of all know the depth of the pockets. To measure the pockets we can best use a gingival sulcus explorer with a bent end, Fig. 3.[1] The bent end of the explorer is placed

[1]Not a "peridental membrane explorer," as we can never explore the peridental membrane.

into the pocket, and keeping the edge on the tooth surface, it is gently moved down to the bottom of the pocket. That part of the gingiva which is occlusally from the bottom of the pocket is the free gingiva. This part of the gum can be considered as useless for any support of the tooth and it should be eliminated.

After administration of a local infiltrative anesthetic into the buccal and lingual mucous folds (and not into the gingivae) the pockets are again measured, and if needed, their depths can be marked on the outer surface of the gingiva by pricking it with an explorer.

It has often been asked whether or not conductive anesthesia should be used. Local infiltration is usually satisfactory, but there is no objection to the application of conductive anesthesia other than the fact that it seems to be too drastic a measure for a relatively minor operation.

A pair of knives and scissors have given me the best service in performing this operation, Fig. 4.

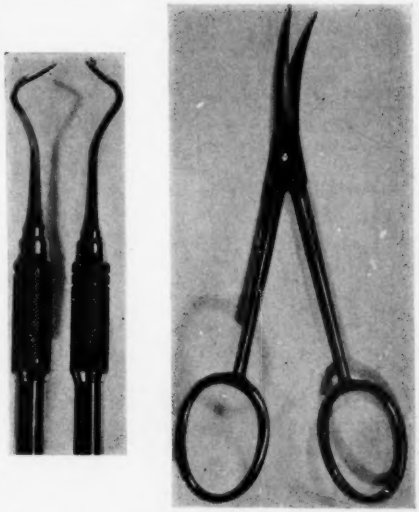

Figure 4—A pair of knives and scissors for performing gingivectomy. The knives have a contra-angle and are sharp on both ends. Both scissors and knives must be extremely sharp.

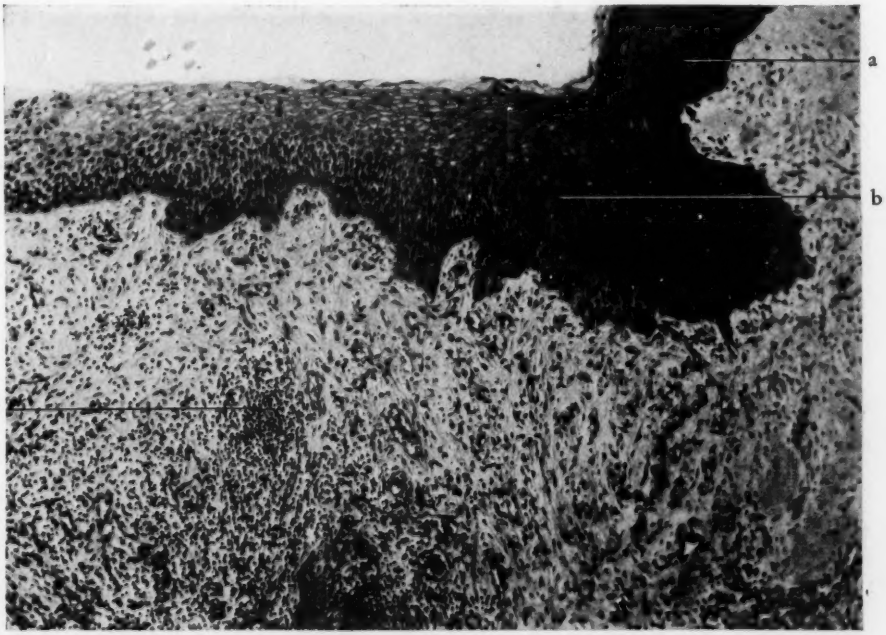

Figure 5—Gingivectomy was performed eight days previous to obtaining this biopsy specimen. During the eight days $AgNo_3$ was applied three times. The healing of the surface is progressing satisfactorily. (a) old epithelium; (b) new epithelium which grew during the eight days; (c) subepithelial connective tissue somewhat inflamed.

The free gingivae must be removed from around the tooth, following the bottom of the sulcus but not in a straight line, rather follow the outline of the depth of the pocket. Single pockets can be eliminated this way, or an entire quarter of the mouth can be operated on at one time. If gingivectomy is to be performed in the entire mouth, one can divide the field into four or six sections and the operation can be done at intervals of two or more days. The sooner the entire operation is completed, the less the amount of time consumed and the easier will be the post-operative treatment. The wound surfaces and edges should be smooth and clean; the tissue should not be traumatized.

After the operation is performed the bleeding must be stopped. This can be done best with some small pieces of gauze dipped into 1/1000 solution of epinephrin or by packing small pieces of gauze, dipped into 8% zinc chloride, into the spaces between the teeth. These packs should stay from 5 to 10 minutes, or may be changed once in between. After the bleeding has stopped, a zinc oxide-eugenol pack[1] is placed on the wound surface. This zinc oxide pack should be spatulated, with a stiff spatula using a waxed paper pad, until it is a stiff mix. It should be stiff enough to enable one to take it between the fingers and form it into a roll—then it can be placed where necessary. It is best to push this pack between the teeth with the help of the patient's lip or cheek. The pack, penetrating from the labial side to the lingual side between the teeth and below the contact points, will be sufficiently anchored to stay in place.

In experimental investigation during the past year we were able to observe that the healing of the gum wound progresses best if the pack is changed at least three times within ten to fourteen days. If it is left on the wound for twelve to fourteen days, as several operators advocate, the healing is delayed. Figs. 4 and 5, show the healing process when the pack has been changed and in continuous action. When the pack was changed the surface of the newly

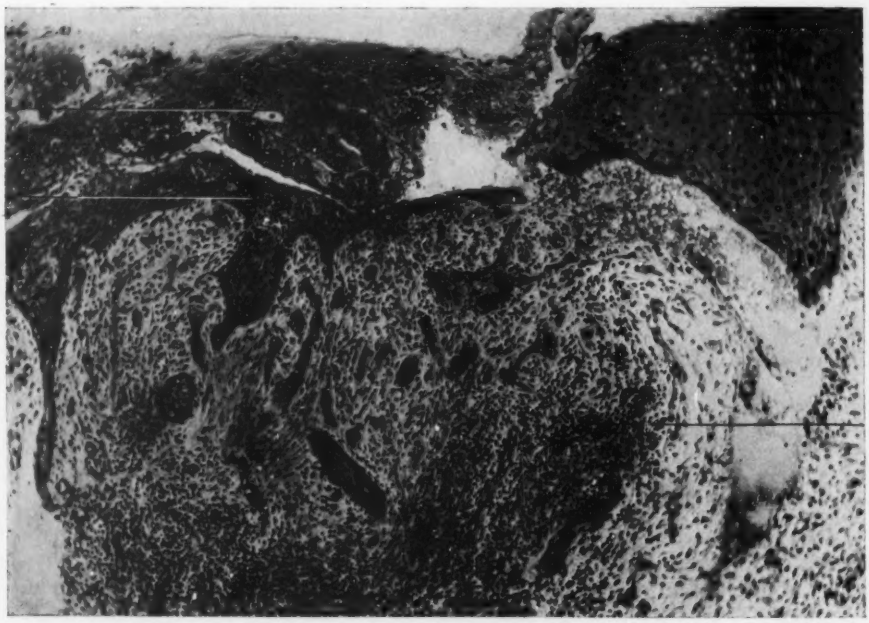

Figure 6—Gingivectomy was performed nine days previous to obtaining this biopsy specimen. The wound surface was covered during the entire nine days with a zinc oxide-eugenol pack. Above the wound surface remnants of the partly degenerated blood clot (a). The wound is unclean. The new epithelium (b) is thin and invaded by leukocytes. The subepithelial connective tissue (c) is considerably inflamed. (d) old epithelium.

[1]Formula of the Council on Dental Therapeutics, A. D. A.

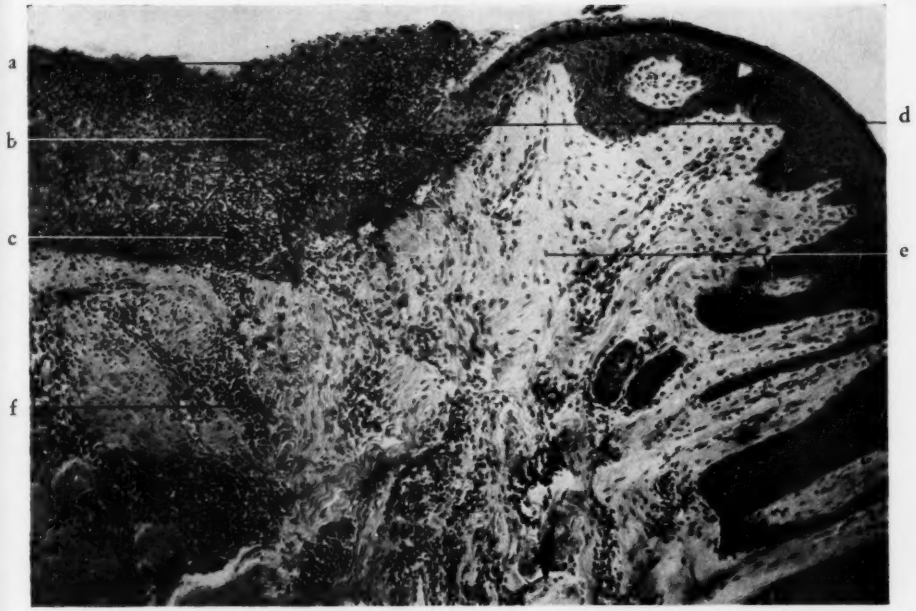

Figure 7—Gingivectomy was performed two days previous to obtaining this biopsy specimen. The blood clot shows three layers; the outer (a) has disintegrated, the middle (b) is invaded by leukocytes and the inner (c) is mostly fibrinous and has become organized. The epithelium (d) has begun to migrate into the blood clot covering the wound. Some of the connective tissue is edematous (e) and another part is still inflamed (f).

formed epithelium was clean and regular, Fig. 5. The subepithelial connective tissue might still have inflammatory cells present but not in abundance. When the pack had not been removed for twelve days, Fig. 6, the surface of the wound was unclean. There are rests of the blood clot, necrotic tissues and bacteria present. The epithelium is irregular, very thin and not continuous. The connective tissue shows an extensive inflammation, which is most probably due to the action of toxins from the unclean wound surface upon the underlying tissues.

This investigation is still in progress and is being carried out in coöperation with Dr. E. Archer as a graduate problem. The results obtained up to now justify our practical conclusions—to change the pack every two to four days until the healing is completed, which will be in about fourteen days.

Every time the pack is changed the use of some drug, such as silver nitrate, zinc chloride or trichloracetic acid, is indicated. The action of these drugs is to coagulate the surface. This should be done to eliminate the overgrowth of granulation tissue and to stimulate the growth of healthy epithelium on the surface of the exposed connective tissue. Fig. 7, shows a gingival specimen two days after gum resection had been performed. A blood clot covers the exposed connective tissue wound surface. Upon close investigation we can observe that this blood clot consists of three layers. The inner layer shows a network of fibrin invaded by leukocytes. This layer as serial investigations

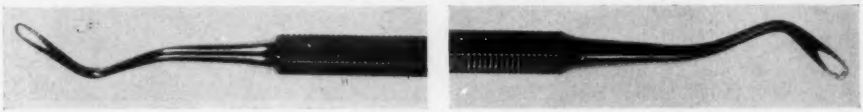

Figure 8—Silver nitrate instrument. The loop-ends are heated and $AgNo_3$ crystals picked up and melted into the loop.

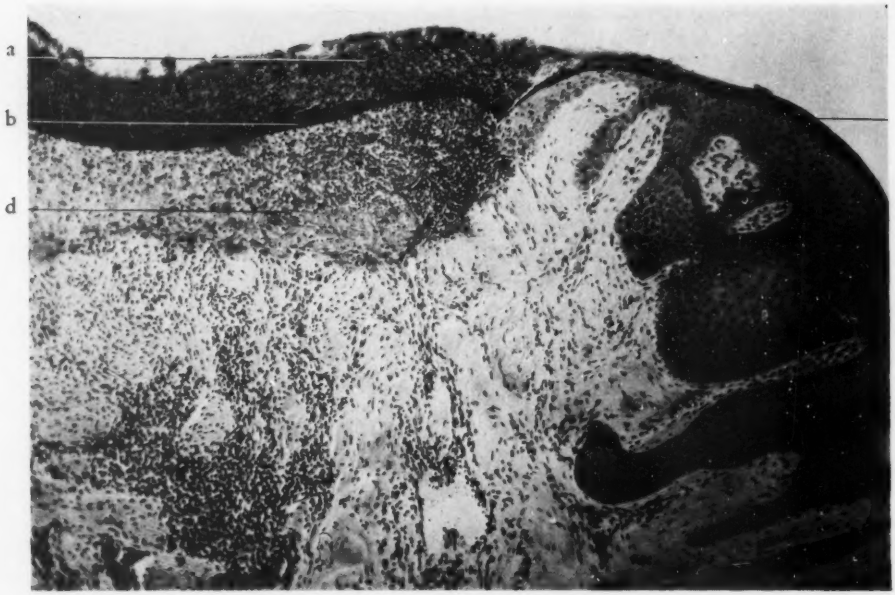

Figure 9—The same specimen as in Figure 7, but silver nitrate applied to the wound surface. The silver nitrate penetrates the blood clot about one-third (a). The innermost layer of the silver deposit (b) is heaviest. This deposit limits further penetration. The healthy epithelium keeps the silver nitrate deposit on the surface, (c). d: fibrinous part of the blood clot.

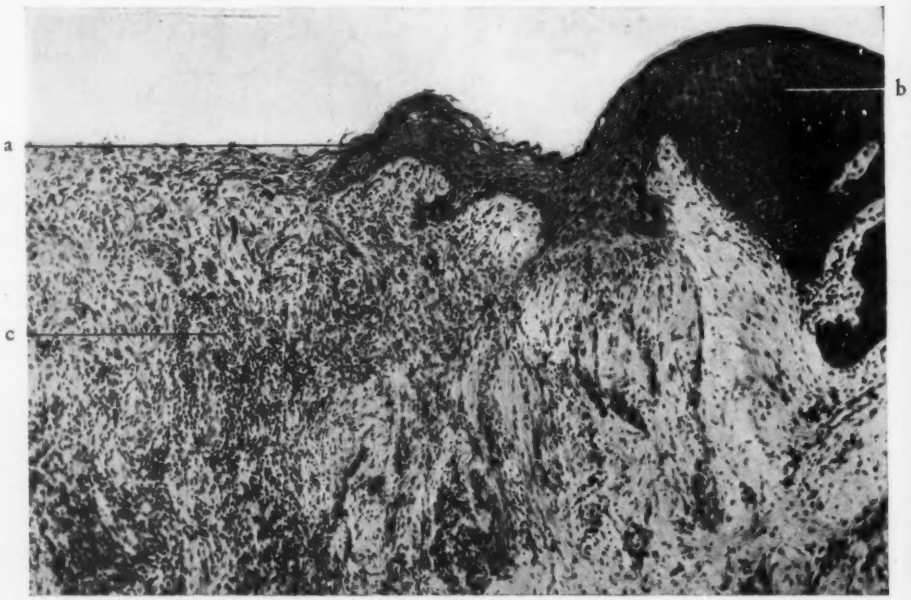

Figure 10—Specimen from the same individual as in Figures 7 and 9, but five days after gingivectomy and three silver nitrate applications. The new epithelium (a) migrates on a clean wound surface and is in continuous connection with the old epithelium (b). The connective tissue is still inflamed (c).

have shown, becomes organized and is transformed into a part of the tissue. In the middle layer of the clot a larger number of leukocytes accumulate, while in the outer layer the cellular elements show signs of disintegration. This outer layer becomes necrotic due to the direct action of bacteria and their toxins. It is reasonable to believe that the accumulation of leukocytes in the middle layer is due to the necrosis of the outer surface. This leukocytic accumulation is a cellular demarcation and in this way the necrotic outer layer is eliminated. The epithelium at the wound edge started to grow into the blood clot. The epithelium is not growing on the surface of the clot, nor below it, but into it. It is growing in the layer which separates the outer from the inner layer—the layer of demarcation above the fibrin network which becomes organized.

If we apply silver nitrate, in the form of crystals melted to an instrument, Fig. 8, to the gum surface the silver nitrate will penetrate into the blood clot to a certain distance, usually about half way, as shown in Fig. 9. On the surface of the normal epithelium the silver nitrate scarcely penetrates at all, but in the blood clot it penetrates from five to ten cell layers in depth. The innermost layer of silver nitrate precipitate is the heaviest; the silver is deposited here in all spaces between the cells as well as in the cells. The day after the application of the silver nitrate, this silver protein precipitate can be removed easily, and is usually washed away by using a spray to clean the mouth. If zinc chloride and trichloracetic acid penetrate deeper into the tissues, they are not so sharply limited, and their precipitate is not so easily removed. After the precipitate is removed the drug can be applied again. The effect of the 10% zinc chloride or 10% trichloracetic acid is similar to that of silver nitrate, but the action of silver nitrate is cleaner and more definite than anything else we have tried. After two changes of the pack and the action of the silver nitrate, the surface of the wound is clean, Fig. 10. No necrotic tissue hinders the complete healing of the surface as it is covered with healthy epithelium and there is a gradual diminution of the inflammation in the connective tissue.

After about two applications of the zinc oxide-eugenol dressing, a paraffine or paraffine-beeswax pack can be used. This is easier to apply and easier to remove and it helps to keep the wound protected. After twelve to fourteen days a pack is not necessary. Silver nitrate, zinc chloride or trichloracetic acid can be further used whenever red spots appear in any particular region. At this time proper home care constituting massage, interdental stimulation, use of toothpicks, dental floss and pipe cleaners should be started, and the success of the entire operation depends on the vigorous execution of the proper measures and regular check-ups.

Several requests have been received for copies of the January and July, 1933 issues of the Journal of Periodontology. As our supply is exhausted we would appreciate hearing from anyone who is willing to dispose of his copies of these numbers. Kindly communicate with the Editor, 555 West Maple Avenue, Birmingham, Michigan.

Case of Gingivoma, or Elephantiasis of the Gingiva*

By EDWARD L. BALL, D. D. S., *Cincinnati, Ohio*

A WELL developed school boy, fourteen years old, with excessive hyperplasia of the gingiva, came to me for examination and treatment in July, 1939.

The patient gave a history of having had a troublesome condition of the gums for many years.

Past history showed that he was a patient in the Good Samaritan Hospital, Cincinnati, in April, 1928. He was three years old at that time and the chief complaint was "Bad condition of the gums." The pediatrician's diagnosis was "Keloid of the gums." The record reports head, neck, chest, heart, abdomen, and extremities normal. Blood Wasserman negative. Blood calcium normal. Tuberculin test negative. Complete blood and urinalysis normal. The child stayed in the hospital three days; no treatment reported. At the age of five the child had his tonsils and adenoids removed at the hospital. Recovery uneventful. Between the ages of three and five years a dentist removed the hypertrophied tissue and all of the deciduous teeth with the exception of the left upper cuspid.

Figure 1.—Front view. J. S., age 14. Note protrusion of lips.

The tissue gradually hypertrophied again but due to dissension in the family and possibly for economic reasons, nothing was done about the oral condition for nine years. A dental school nurse called a visiting dentist's attention to the markedly swollen gums in May, 1939.

The boy was then 14 years old. His general physical examination showed him to be normal. Complete blood examination and urinalysis normal. Wasserman negative.

Clinical examination of the mouth revealed a marked hypertrophy of the gums with the permanent teeth erupting in a few places. Dental radiograms showed all of the permanent teeth present and also the remaining deciduous cuspid.

This condition has been described under a variety of names, "fibroma of the gums," "gingivoma," "elephantiasis of the gingiva."

Specimens of the gingival tissue have been examined by seven leading pathologists including Dr. William German of the Good Samaritan Hospital, Cincinnati, who reported his own views of the case and also an analysis of the opinions expressed by the others.

Dr. German's Report is as follows:

Histological sections of gingival tissue show hypertrophy of epithelium. This shows mild hyperkeratosis and the production of long rete pegs which extend down into the underlying stoma. The principal lesion is in the stoma and this shows a tremendous

*Read before the American Academy of Periodontology, at Cleveland, Ohio, September 7, 1940.

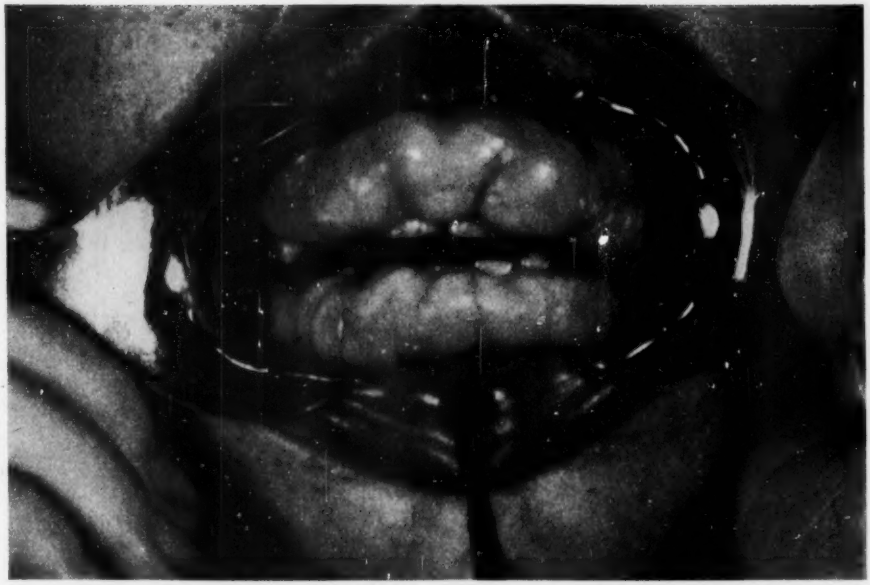

Figure 2.—Anterior view: Tissue unusually hard.

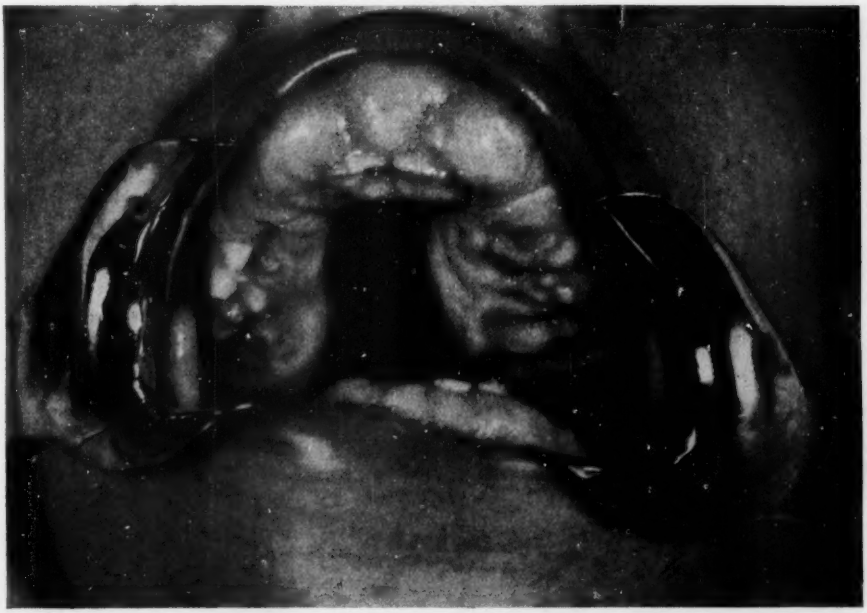

Figure 3. Lingual view: Note width of palate. At times the lingual tissue became markedly swollen, practically closing the palate.

Figure 4. One month after operating the upper jaw.

Figure 5. Case after surgery.

Figure 6—Lingual view. Comparing width of the palate before and after surgery.

hypertrophy of collagenous connective tissue. The collagen bundles are hypertrophied. The tissue shows a high degree of differentiation, though there are a few young fibro-blasts. The connective tissue bundles show the usual degree of interlacing, except those near the surface which project in the form of large finger-like papillae into the overlying epithelium. There are a few collections of inflammatory cells, this due undoubtedly to a mild inflammatory reaction to food debris between the overgrown gingival tissue and teeth. Hortega silver and gold stains show absence of elastic tissue and reticulum. No nervous elements found. The older collagen shows some degeneration as revealed by its becoming slightly argentophil.

COMMENT

The sections were reviewed by seven pathologists. Upon several points there was unanimity of opinion. These points may be listed as follows:

1. That the lesion involves the gingival connective tissue.

2. That there is marked hypertrophy of overgrowth of this connective tissue.

3. That it is a benign lesion from the standpoint of tumor diagnosis.

4. That it is covered by squamous epithelium which likewise shows no neoplasia.

5. That there are few areas showing inflammation and that these areas are associated with blood vessels.

Of the seven pathologists who reviewed the sections, three have classed the lesion as a neoplasm, as judged by their diagnosis of fibroma. From the opinions expressed by the other four, it can be inferred that it is not a neoplasm, but that it represents a tissue hypertrophy or overgrowth. One report suggested as etiology an endocrine disturbance or developmental anomaly of mesenchymal tissue growth.

In my opinion this is not a neoplasm but a connective tissue hypertrophy similar to keloid. Connective tissue reaction to form keloid is a constitutional phenomenon occurring in certain individuals, families or races. It would be interesting to observe if there is any attempt at scar hypertrophy or keloid formation in the tonsillar fossae, as the result of tonsillectomy. It will probably recur, though radiation therapy may retard its progress . . .

William German, M. D.

In August and September of 1939 the case was operated. Under block anesthesia the tissue was removed on the buccal, labial and lingual sides of all the upper and lower teeth. This was accomplished in five operations, the tissue being removed down to the periosteum between all of the teeth and well beyond the alveolar margin. This surgical procedure was similar to the surgical treatment of periodontoclasia, however, decidedly more radical. After the soft tissue was removed the edges were cauterized and the bone and tissue covered with a surgical dressing (Ward's) which consisted of a combination of zinc oxide and eugenol paste. This medicated cement hardens, protecting the tender tissue, keeping the area clean, and reducing the after pain to a minimum. The dressing was removed every two or three days, the areas irrigated, and the edges touched with full strength silver nitrate. This was repeated for two weeks after each operation.

The patient seems perfectly well and has gained thirty pounds.

Six months after the case was operated, deep X-ray treatments were started by Drs. Doughty, Bader, and Reineke, and their report is as follows:

We finished with a second series of deep X-ray treatments to your patient, Jack S. on July 19th. He has now had a total dosage of 4000 r units. We are of the opinion that this will complete the X-ray treatments, however, we have asked him to return in about one month for observation. If at some later period you notice any definite change, we will then consider the advisability of more treatment.—Signed Drs. Doughty, Bader and Reineke.

May I express my deepest appreciation to the following pathologists for their reports: Drs. Kronfeld, Orban, Becks, Sinclair, Kitchen, German, Mann, Kotangi and Ash, to Dr. Frederick Black, orthodontist, for his impressions and models, and to Drs. Doughty, Bader, and Reineke for their X-ray services.

814 Doctors Building.

* * *

DISCUSSION OF THE "CASE OF GINGIVOMA, OR ELEPHANTIASIS OF THE GINGIVA," REPORTED BY EDWARD L. BALL

By B. ORBAN, M.D., D.D.S., *Chicago, Illinois**

FROM the specimen sent to me by Dr. Ball, I can confirm the description given by Dr. William German. The hypertrophy of the gingivae is due to extensive overgrowth of the subepithelial connective tissue. The interlocking fiber bundles of collagenous nature are enlarged, as seen in Fig. 1. The cellular elements seem to be rather reduced. Only around some capillaries can we see accumulations of inflammatory cells. The epithelium is well keratinized on the surface, but the prickle cell layer is largely reduced.

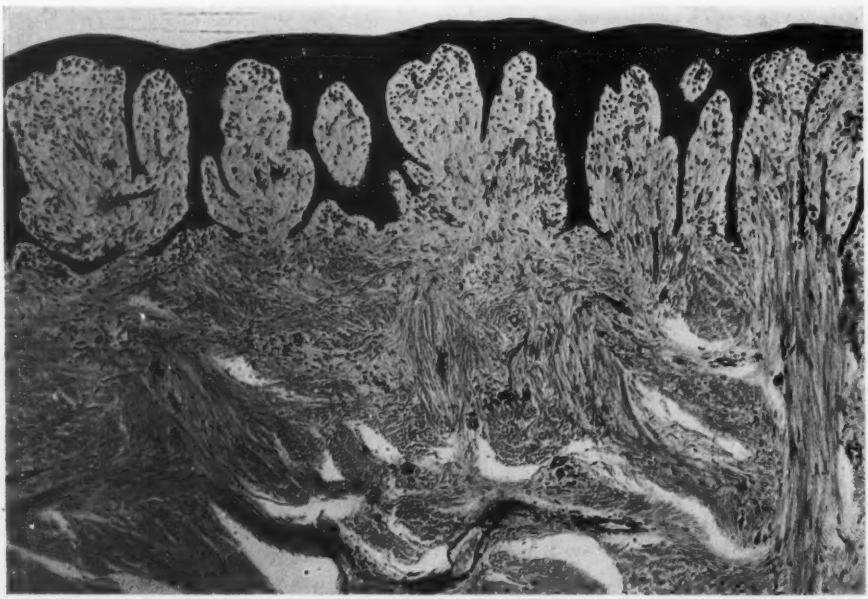

Figure 1—Atrophic epithelium (a) covering the hypertrophic connective tissue (b) long narrow pegs of the epithelium penetrate into the connective tissue.

*Foundation of Dental Research of the Chicago College of Dental Surgery.

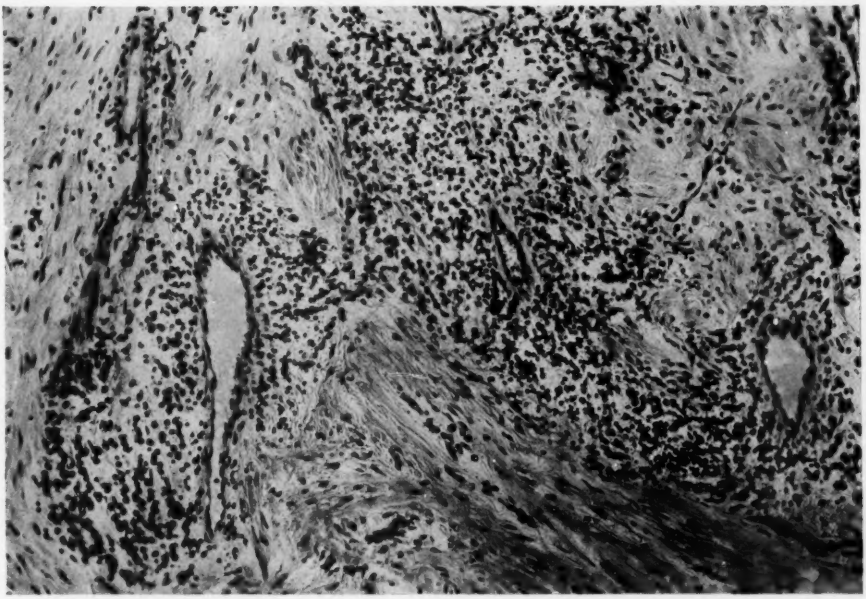

Figure 2—Round cell infiltration around blood vessels. The round cells are mostly plasma cells, and some lymphocytes.

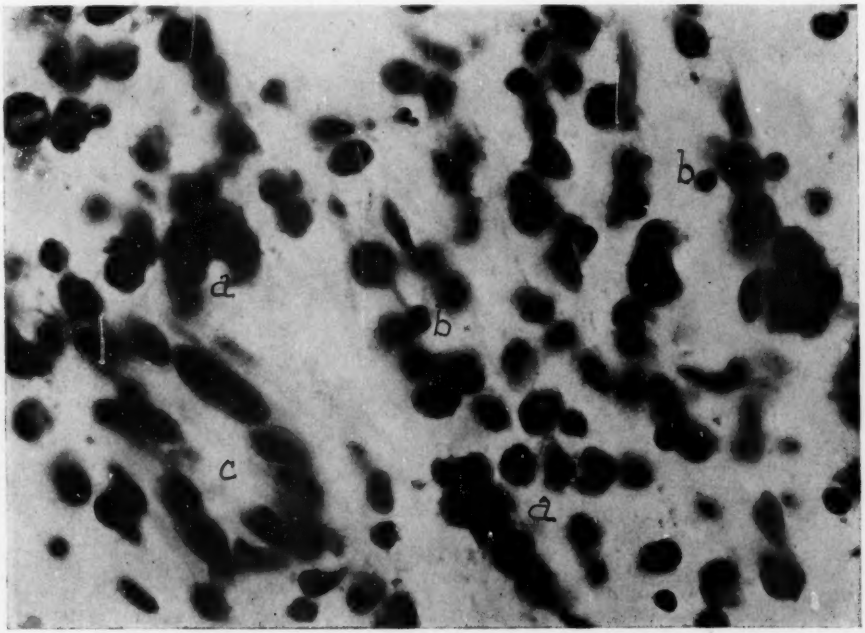

Figure 3—High magnification of the plasma cells (a) and lymphocytes (b) around capillaries (c).

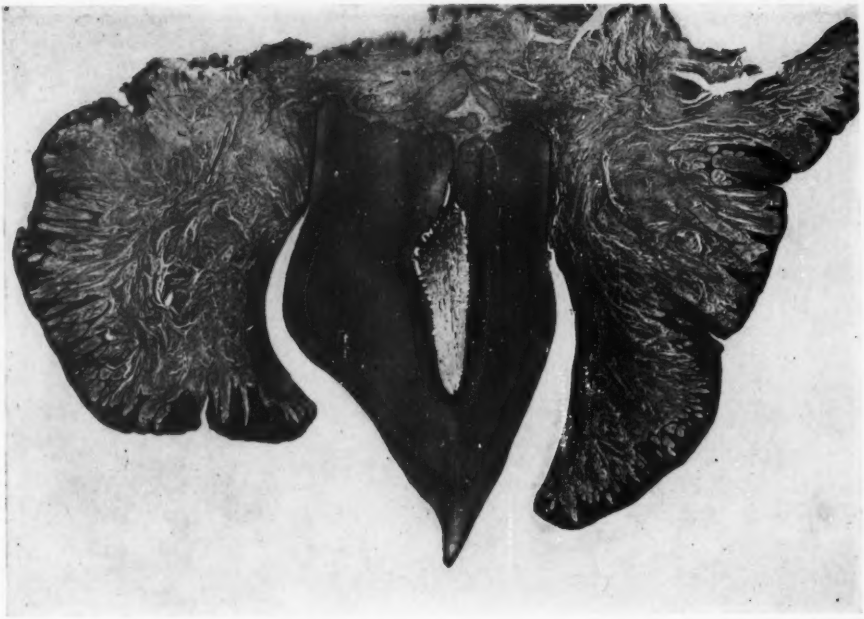

Figure 4—Deciduous upper cuspid, with hypertrophic gingivae. Root resorbed to gingival third. Bone formed above the root end.

Figure 5—Root end of deciduous cuspid (a). (This plate is reversed, top to bottom—Ed.) Bone (b) under the resorbed root end showing giant cells resorbing the bone. On the surface of the resorbed root end bone is laid down on some areas, while resorption is going on at others.

The long peg-shaped processes of the epithelium show somewhat more cells in the stratum spinosum.

This atrophy of the epithelium is most probably due to pressure from the overgrowing connective tissue. While most of the hypertrophic tissue is free from inflammation, there are regions quite severely infiltrated. Fig. 2, shows one of these areas. Round cells are accumulating around blood vessels. As can be seen in a still higher magnification, Fig. 3, most of these cells are plasma cells, but some of them are lymphocytes. These round cell infiltrations are close to the gingival crevice or to the surface where deeper grooves develop in the hypertrophic gingivae. The formation of deep epithelial fissures and grooves, being a characteristic in hypertrophic gingivitis, was lately described by Gottlieb. The entire character of this hypertrophic disorder is not inflammatory, in spite of the presence of several inflammatory cell accumulations. These cells are relatively few when compared with the tissue growth present.

The deciduous upper left cuspid was removed, together with the hypertrophic gingivae, by Dr. Ball. Fig. 4, shows this tooth with its surroundings. The root is resorbed to the gingival third. At the base there are some bone trabeculae present, which indicates alternating resorption and repair periods in the process of deciduous tooth shedding. The higher magnification of this region is shown in Fig. 5. Resorption of bone is taking place in several places and new formation of bone in other places. Bone is also deposited on several areas of the resorbed root and resorption line is seen in others. This shows that activity was present at the time the tooth was extracted, and in time this tooth would have been cast out due to loss of its entire root.

In summarizing, I can state that the hypertrophy of this gingiva is due to growth of the collagenous fiber masses of the subepithelial connective tissue. Inflammation is not a primary factor in this disturbance. Inflammatory cells are found surrounding blood vessels but they are rather scarce and close to the surface. The inflammation seems to be due to factors of local character, such as irritation in the gingival crevice and fissure formations in the hypertrophic gingivae.

The epithelium covering the hypertrophic connective tissue is rather atrophic and shows a relatively thick layer of keratin on the surface and it sends long and narrow pegs into the connective tissue.

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Twenty-Eighth Annual Meeting

AMERICAN ACADEMY OF PERIODONTOLOGY

HOUSTON, TEXAS, OCTOBER 23, 24, 25, 1941

THURSDAY

9:00 A. M.—Registration

9:30

First Session of the Twenty-Eighth Annual Meeting.

Isador Hirschfeld, President.

REPORTS OF OFFICERS AND COMMITTEES:

Secretary-Treasurer. Raymond E. Johnson.

Committee on Program. James A. Sinclair.

Committee on Clinics. Hunter S. Allen.

Committee on General Arrangements. Walter H. Scherer.

Committee on Scientific Investigation. Balint Orban.

Committee on Nomenclature. Harry Lyons.

Committee on Publication. H. J. Leonard.

Committee on Ethics. Edward B. Spalding.

Committee on Credentials. W. A. Beatty.

Special Committee Reports by: Dickson G. Bell, Allison G. James, Leo F. Marre, Harold J. Leonard, Russell A. Sand, Samuel R. Parks, Juanita Wade, Haidee Weeks, Edgar D. Coolidge, Grace Rogers Spalding.

11:00

Presidential Address. Isador Hirschfeld.

2:00 P. M.

Principles of treatment evolved from experience and observation. Walter Leabo, Shreveport, La.

3:00

Oral manifestations of vitamin deficiencies. George S. McReynolds, M. D., Galveston.

4:00

Standard norms of salivary composition. William W. Wainwright, San Francisco.

FRIDAY

8:00 A. M.—Past-Presidents' Breakfast.

9:00

Report of past-presidents' meeting. Olin Kirkland, Chairman.

9:15

The periodontist in Eastern Asia. R. Gordon Agnew, Toronto, Can.

9:50

The effect of vitamin B filtrate factor on oral tissues of animals. Hermann Becks, San Francisco, Calif.

10:25

Biology of the cementum. B. Gottlieb.

12:30 P. M.

Luncheon—Academy Round Table. Allison G. James, and C. J. Caraballo, Co-Chairmen.

1. Is periodontia more the result than the cause of systemic disease. Russell A. Sand, *Leader.*
2. The role of nutrition in the treatment and the prevention of periodontal disease. R. Gordon Agnew, *Leader.*
3. What results can the periodontist expect from thyroid medication to patients with hypothyroidism. Hermann Becks, *Leader.*
4. Vincent's infection complication in periodontoclasia and early treatment of the same. J. O. Hall, *Leader.*
5. Drugs in periodontal therapy. R. Dean Creamer, *Leader.*
6. Indication for types of operative procedure in periodontal treatment. Justin D. Towner, *Leader.*
7. Instruction for proper home care in periodontal cases. Robert L. Dement, *Leader.*
8. The relative value of mouth rehabilitation and periodontal treatment in mutilated mouths. W. F. Andes, *Leader.*
9. Suggestions for promoting research in periodontology, and the possibility of research grants to accredited institutions or individuals. Dickson G. Bell, *Leader.*
10. Ladies' Group. Mrs. Russell A. Sand, Presiding. (Subject to be announced.)

2:30

Clinics. Hunter S. Allen, *Chairman.*

Oral manifestation of an undetermined fungus infection. Walter H. Scherer, Houston, Texas. Assisted by E. W. Bertner, M.D., and Herbert H. Harris, M.D., Houston, Texas.

Optimum occlusion. J. C. Westbrook, Jr., Birmingham, Ala.

How shall I manage periodontal disease. Thomas E. Prosser, Jr., East St. Louis, Ill.

The clinical importance of an exact diagnosis of loose teeth. Emmerich Kotanyi, Houston, Texas.

Periodontal disease illustrated by radiographs, models, and kodachrome photographs. Rowe Smith, Texarkana, Ark.

Simple and practical aids to the periodontist. W. A. Beatty, Houston, Texas.

Visual education for the periodontal patient. E. R. Romine, Detroit, Mich.

Mouth rehabilitation in relation to periodontia. H. J. Leonard, New York City.

Saving the lower anterior teeth after periodontal treatment by properly constructed partial denture. W. J. Healey, New Orleans, La.

Practical care of full mouth reconstruction emphasizing the preservation of the periodontium. F. Raymond Garvey, St. Paul.

The third molar as an etiologic factor in periodontoclasia. William B. Cockroft, Memphis, Tenn.

7:30

Annual Banquet. Houston Country Club.

SATURDAY

9:15 A. M.

Incidents of Office Practice.

A case of periodontoclasia treated surgically. B. F. Thielen, Paris, Texas.

Osteomyelitis from a periodontal pocket. R. Dean Creamer, Phoenix, Ariz.

A case history showing dietary influence on certain types of periodontal lesions. J. O. Hall, Waco, Texas.

Acute monocytic leukemia with pronounced gingival involvement. Virgil Loeb, St. Louis, Mo.

Vincent's infection. Ray Gill, San Francisco, Calif.

Differential diagnosis between lateral abscess and osteomyelitis. Balint Orban, Chicago, Ill.

Padgett's disease of the mandible. (Osteitis deformans.) J. B. Freedland, Charlotte, N. C.

Root amputation. Robert L. Dement, Atlanta, Ga.

Case report. L. A. Neil, El Paso, Texas.

A desperate case and its solution. Harold J. Leonard, New York City.

Diseases of the oral mucosa. Thomas J. Cook, Philadelphia, Penna. (Illustrated with colored motion pictures.)

SUPPURATIVE INFLAMMATION OF THE GUMS

(Continued from Page 82)

Fourth and last stage. Where the disease has swept away all the alveoli and much of the gum, the tooth being held in place by the conversion of the peridental membrane of the apex of the root into a tough ligamentous attachment.

The first and second stage is seldom noticed—only when it has reached the third stage does it challenge the attention of the practitioner; then to be treated by astringent washes or styptic tooth powders, or by the more heroic treatment of slitting the gums between the teeth, down to the bone, with a thin thumb lancet, only to relieve the turgid gums temporarily by copious bleeding. The patient is informed that he has scurvy of the gums, or bone disease, or old age disease, though the subject be not thirty years of age; or that the disease is hereditary or constitutional; or the patient is of a scrofulous diathesis.

So much for the old pathology and treatment. What saith the new? I plead a general denial of the old pathology and treatment, and offer the following point in proof of its falsity.

Present the worst case possible, one of those described in the first pages of this paper, and it shall make full and complete answer. Extract every tooth in said case and the patient will rapidly recover his health; his jaws and gums will immediately show an improved appearance; the gum tissue will change from a dark red and swollen condition to a light pink and healthy color, and the whole mouth in due time will present as healthy a base for an artificial denture as was ever seen. Now, if it is a born disease, or hereditary, this sudden loss of the teeth should work no cure, but the disease should and would march straight forward until all the processes of both jaws were annihilated, if so be the patient lived so long. Not so, however. The teeth being lost, the disease is arrested, active and suppurative inflammation and absorption of the parts subside into a curative and healthy action. New bone cells are deposited into the remaining alveoli until the apex or ridge of the jaw is rounded over and health reigns where a disgusting and wasting disease lately committed its ravages, and what is more and better, health, vigor and happiness light up the eye and crown the brow where sat inanition and gaunt despair.

This is no fancy picture, but a slight outline of the true portrait. I plead, secondly, an affirmation, and adduce the proof that the teeth themselves, with their accumulated accretions and roughened surfaces, from whatever source derived, are the exciting cause of the disease under consideration. The teeth in perfect polish and cleanliness, at and under the margin of the gums, whether of animals or man, produce no inflamed action in that tissue. It can be artificially produced, however, by inserting a foreign body into or beneath its substance. If then diseased action can be set up by a foreign body, artificially introduced, it can be arrested and cured by withdrawing the same. And, therefore, if the tooth becomes an extraneous body by reason of the accretions and concretions upon it, near and under the free margin of the gum, and inflammation ensues, as it certainly will, the true prophylactic and pathologic treatment surely would be to thoroughly and carefully remove said concretion, tartar or roughness—polish the tooth and let nature take care of the rest . . .

I have pointed out the treatment of the disease in the first stage.

The treatment of the second stage is the same, only being careful to reach the extreme limits of the diseased action, breaking up the diseased tissue and removing every particle of tartar from the tooth, and necrosed bone from edge or margin of process.

The third stage presents greater difficulties from the greater depth of the active line of disease, and demands a firm and skilled hand, a delicate and nice touch, and, I might add, the transfer of the sense of sight to the finger ends. The sense of touch must be so trained and cultivated that all foreign bodies can be readily distinguished from tooth substance—live bone from necrosed bone—healthy from diseased tissue. This manipulation cannot be attained at once, but time and practice, with close and earnest study, will qualify and school the hand, and embolden the true and sensitive mind to achieve success in the treatment of this third stage.

Of the treatment of the fourth and last stage, little can be said, except that the loss of the tooth or teeth is inevitable. The tooth is held in place by a cartilaginous attachment or condition of the inverting root membrane, which holds it in place as a buoy is held in the water. The only alternative is extraction . . . and nature refuses to reconstruct the socket anew . . .

There is a point, however, in the third stage, where one or more teeth begin to grow loose, that restoration to health and firmness, is not only possible, but quite certain, provided the operator is so self-possessed as to *know when* he has reached the utmost limit of the disease. If he fail to make a clean operation, that is, to go fully to the line of healthy bone, and to remove all necrosed bone and all foreign accumulations on the tooth, the disease is sure to show the fact instanter. If a speck of tartar, not larger than a small grain of gunpowder, be overlooked, the gum over it will manifest the fact by a reddened patch of the tissue several lines greater than the tartar underneath. We will assume, then, that operation is thoroughly performed. What then? Why this, surely, new bone cells are deposited in the crater-like depression in which the tooth stands, and calcifies around the tooth and makes it firm and strong in position. The gum firmly grasps the root above this new-formed bone, and presents a perfectly healthy appearance.

Some mouths present all four stages of the disease at the same time, whilst others, the younger patients, present only the first or second. So far from being a disease of old age, cases are numerous at seventeen, and even fourteen years. These, however, are in the first stage, and blessed are the eyes of that man who recognizes it and applies the remedial treatment thus early. No medicinal treatment is needed, further than an astringent wash and myrrh tincture, full strength, touched to the gum with the end of the finger, is best of all.

[NOTE:—*The above copy of Dr. Riggs' paper ought to serve as a means to correct the statement made by some writers that Dr. Riggs used radical surgical methods in the treatment of his cases.*—ED.]

Editorials

The Cloud Has a Silver Lining

ACCORDING to the tradition of clouds, the one hanging over dentistry, altho cumulative and menacing at times, has a silver lining. The latter is comfortingly described by the Editor of the *Journal of the American Dental Association* in the June, 1941, issue. The Editorial is closed with the following assurance:

> The future of dentistry is indeed bright, with a broadening horizon that constantly urges and demands the broadening of the scope of our knowledge.

We cannot but wonder if this was a whistle in the dark, since we are daily conscious of the uncertain future of dentistry as we now know it. We are of the opinion that we will look back upon this period as the *golden age* of dentistry.

These are times when it is advantageous to recognize unpleasant truths and conditions in order to study and evaluate their significance. It is generally known that the dental profession, already weak in personnel, is being still further weakened in this respect by increasing educational standards and the needs of the Army; the dental schools are crying for more matriculants of better quality; in spite of this need, women are being discouraged about studying dentistry by several of our dental schools refusing them admittance even though they are especially well suited for various phases of dental practice.

It is common knowledge that instead of less skill being required of dentists, since there are higher educational standards for them, more skill is needed to apply the broader knowledge gained. Radiograms of one million mouths exhibiting American dentistry would testify to the sober truth that the average dentist is not skillful enough.

The propagandizing of full denture prosthesis has resulted in a new fashion in teeth—extraction of the natural and the wearing of artificial. Extraction of sound teeth is on the increase as a time saver for the dentist and because it is an economically sound and easy procedure for him; it is not always so for the patient. We are disheartened to see this custom growing since too often twenty or more teeth are extracted for patients before middle age, when the removal of a few, with proper treatment of the remaining ones, would have eliminated all infection from the mouth. It is not difficult to remove teeth and much of the work involved in their replacement with artificial substitutes is done by the laboratory technician. This will be even more common practice if dentistry becomes socialized.

We have a shortage of dentists and increasing educational requirements for those entering the profession. Dental caries is on the increase. Many dental pulps die under costly gold inlays on account of insufficient protection, extensive cavity preparation, or careless occlusal adjustment. Periodontal disease is still allowed to progress in the mouths of dental patients until it has reached an advanced or incurable stage when the teeth are hurriedly extracted in an attempt to correct the injury to patients' health, often extending over many years. Improvement is needed in our methods of *dental practice* as the above mentioned facts suggest.

The public does not fully appreciate the importance of oral health nor the

educational, common sense, and skill requirements of the dentist for satisfactory dental diagnosis and dental health service.

The imminence of the socialization of dentistry has been temporarily obscured from us by the war but it remains a serious menace to the advancement of our highly specialized profession. More dental health service is needed and something effectual must be done about that, but until the individual citizen becomes more conscious of the value of his teeth and assumes greater personal responsibility with regard to their care, the efforts of dentistry are largely scattered to the four winds and much of dentists' refined skill and higher education wasted.

These frankly stated but constructive criticisms indicate in part what goes to make up the cloud over us and we are inclined to wonder just how bright is the future of dentistry.

Nomenclature Associated With Periodontology

IT WILL continue to be the policy of the *Journal of Periodontology* to publish reports, discussions, and papers which will aid in clarifying and in settling permanently the nomenclature relating to the supporting tissues of the teeth, their hygiene, and their diseases.

There is a group of dentists who contend that the selection of correct terms will be quite simple after we have collected sufficient scientific evidence to enable us to understand fully the development and function of the periodontal tissues, and the progress of disease in them. This contention sounds reasonable and, therefore, attention is called particularly to the article by Dr. Weinmann, p. 71, and especially to the illustrations. If the interpretation of these microphotographs is correct, many of us will need to readjust our ideas relating to the progress of gingivitis, and also, perhaps, with regard to one or two pet terms. There is no question but that we must first understand a disease thoroughly and accurately in order to correctly name it.

In the meantime we make a plea for consistency in the use of terms associated with periodontology. Many readers of this Journal are periodontists who study the science of periodontology, practice periodontia, and diagnose and treat periodontal diseases. At the very least these few terms should be used correctly. It would also improve greatly the quality of the literature pertaining to periodontia and periodontology if its contributors would avoid using the term pyorrhea alveolaris, especially when describing a condition where there is no suppuration. The term "pyorrhea" means nothing without "alveolaris." Papers are sometimes sent in for publication where a mixture of terms is used throughout them, as for example, paradental, parodontal, peridental, and periodontal are used interchangeably.

A rational consistency in the use of terms relating to the periodontal tissues and their diseases would add stability and interest to periodontology. Dictionaries ought to be used freely by dental writers as well as by dental editors.

A thorough study of the Reports of the Committee on Nomenclature of the American Academy of Periodontology which have been published in this Journal [8(2):88-95. July, 1937 and 12(1):16-18. Jan. 1941] and discussions of these Reports will prepare students of the subject to understand any revision of terms which scientific research may make desirable. *G. R. S.*

ABSTRACTS AND EXTRACTS OF CURRENT DENTAL LITERATURE FOR 1940 (Continued from 12(1) January 1941)

Periodontal Histo-pathology, Periodontal Bacteriology and Clinical Etiology.

(a) Periodontal Histo-pathology

(Continued from 11(2):108. July, 1940)

204—Berman, M.: **Dendritic cells in alveolar gingiva and enamel epithelium; destruction of enamel epithelium by phagocytic cells.** *J. D. Res.* **19**:497-504. Oct., 1940.

Sections of material were taken from 11 month old white male child (post mortem), and stained with hematoxylin and eosin, and with Bodian's activated protargol stain. The sections stained with the latter, showed the presence of dendritic cells throughout the gingival epithelium and in the enamel organ of the erupting teeth. The cells observed here were principally among the outer epithelial cells, while those reported in the literature were in the Malpighian layer. The cells gave a dopa-positive reaction, and seemed to take a lighter stain than similar cells observed in normal tissue obtained in biopsy from light-skinned Caucasians and dark-skinned Negroes.

In some of the sections stained with Bodian's activated protargol, the enamel organ contained large phagocytic cells in the act of engulfing the epithelial cells, and producing large hyalinized areas.

There may be some connection between the presence of dendritic cells and the blood dyscrasias in other diseases; it may offer an explanation for malignant melanoma. Bodian's activated protargol stain might reveal other interesting phenomena, if employed in a study of dental tissues.

—*M. B.*

205—Branchini, C.: **Ueber das Verhalten des retikulo-histozytaeren Systems des Odontons bei kompletter oder abgeschwaechter Karenz von Vitamin C.** Behavior of reticulo-histiocyte system of the teeth in complete or partial lack of vitamin C. *Schw. Mschr. f. Zahnhlk.* **50(4)**:394. 1940.

In the pulp, in the osteoid tissue, in cartilagenous cementum, and in the cementum beads of normal animals can be seen elements with granulocytic capacity, which are classified as histiocytes, pericytes, and fibroblasts; in the pulp, in addition to the above elements, there are the odontoblasts. In animals with complete lack of vitamin C, the parts that show the greatest changes are the pulp and the paradental tissue. The fibroblasts and pericytes exist in great numbers in an intensive granulocytic state; but the histiocytes are most affected by the vitamin C deficiency. The circumference of the histiocytes is greatly increased and they show a round appearance; others show an irregular starry form and range themselves around the hemorrhagic zone and in regions where all the elements of the pulp are destroyed. The mobilization of these elements and their phagocytic power are other signs. The paradental tissues show the same type of elements, but to a lesser degree. In partial vitamin C deficiency, the pulp shows histiocytes in the condition of "wandering cells at rest," and the histiocytes have developed into polyblastic and macrophagic types; they never have the size and appearance seen in full vitamin C deficiency.

—*M. B.*

206—Branchini, C.: **Ueber Veraenderungen beim Odonton und besonders beim Paradentium bei Hypo- und Avitaminose C.** The changes in the tooth and especially in the paradentium in hypo- and a-vitaminosis C. *Schw. Mschr. f. Zahnhlk.* **50(4)**:396. 1940.

Important changes were seen in various parts of the molars and incisors of guinea pigs, in diets lacking in vitamin C. The enamel organ showed diffuse hemorrhage and changes in the cell elements; the greatest changes were in the adamantoblasts which showed a degeneration phenomenon with the

building of small calculary masses, changes which were analagous to those of the odontoblasts. The dentin showed hypoplastic zones and incomplete calcification; the odontoblasts had lost their cylindrical form and regular arrangement, and had laid down a tortuous layer which stained intensely with hematoxylin.

Deficiency signs were very severe in the pulp, and, in some cases, almost the whole pulp was destroyed; frequently, there was an accumulation of amorphous masses of calcium. The phenomena of karyolysis and karyorrhexis were found in the cartilaginous cement, together with abnormal depositions of calcium salts. In the periodontal ligament and alveolar bone, changes were represented by hemorrhage and rarefaction of the bone trabeculae. Deficiency manifestations in the incisors were the same as those in the molars.

—*M. B.*

207—Davidoff, S. M.: **Die neueren Erkentnisse der Paradentoseforschung und ihre Anwertung fuer die Phosphornekrose der Kiefer.** New conceptions of paradentosis study and their evaluation in phosphorous necrosis of the jaw. *Dtsch. Zahn, Mund, und Kieferhlk.* **7**(4):238-52. 1940.

Clinical and experimental studies to determine the mechanism of formation and genesis of phosphorous necrosis of the jaw, contribute information on the causal relationship between phosphorous necrosis of the jaw and the teeth, and throw light on the various factors involved. Why do the symptoms of chronic phosphorous poisoning occur especially in the jaw? The action of phosphorous is not only hematogenous, osteoplastic in nature, but also a cementoposonoblastic action; it is the same on all osteogenic tissue. It causes obliteration of the pulp cavity and root canal, and then heavy apposition of cementum at the roots; this produces an increase in the blood and tissue pressure which, together with disturbances in the nutrition of the bone and tooth tissues, creates the supposition of bone atrophy. We must not overlook the functional-mechanical influence of the paradentium, the great possibility for infection, as well as eventual pathological changes in the pulp, periapical, and paradental tissues. The condensation of the tooth structure contrasts sharply with atrophy of the alveolar bone; then, resorption or destruction of the root of the tooth follows, apparently because the compact non-vital tooth acts as a foreign body. Histological studies of various sections of intestine have shown a chronic enteritis as a concomitant causative factor working for the production of bone changes.

—*M. B.*

208—Helstein, M.: **Patho-histologische Befunde bei Paradentalerkrankung.** Patho-histologic findings in paradental disease. *Schw. Mschr. f. Zahn.* **2**:105. 1940.

The word paradentium is defined as being formed by the gingiva, alveolar process, and the periosteum, which form a whole, with the purpose of supporting the tooth.

There are many etiologic factors involved in the production of paradental diseases. There are mechanical and local factors: malocclusion, traumatic prosthetic appliances, excessive strain on a tooth, and infections of the gingiva. But these exogenous factors are not enough, in themselves, to cause paradentosis, and various endogenous factors are involved: decalcifications, deficiency of vitamins C or D, glandular disorders; there is also a correlation with arthritis, diabetes, Basedow's disease, and some authors consider that all constitutional diseases weaken the paradentium.

Dr. Helstein especially considers, amongst causes of paradentosis, diseases of the skeleton, and first amongst these is Recklinghausen's disease, which is a fibrous osteodystrophy (osteitis fibrosa). Paradentosis constitutes, according to the Geneva school—a slow incipient form of osteodystrophy of Recklinghausen. To investigate this viewpoint, Dr. Helstein studied cases of paradentoses, looking for changes resembling osteitis fibrosa. In 16 cases taken at random (whose case histories are given in full), only two showed histologic modifications agreeing exactly with those of Recklinghausen's disease. To draw definite conclusions, much more material will need to be studied.

—*M. B.*

209—Zerosi, C.: **Das medullaere Knochengewebe des paradentium in seiner feinen Histogenese und Struktur.** Medullary bone of the paradentium in its fine histogenesis and structure. *Schw. Mschr. f. Zahnhlk.* **50**(4):395. 1940.

The author mentions most important histological and histogenetic studies of bones in general, and from this viewpoint he de-

scribes his observations on the fine histogenesis of paradental bone during embryonic life. After comparing this with the development of alveolar bone, (which he has studied formerly in regeneration of paradental bone after pathological dental processes), he describes the structure of adult alveolar bone, and illustrates its histologic characteristics.

—*M. B.*

(b) Periodontal Bacteriology

(Continued from 11(2):110. July, 1940)

92—Brewer, J. H.: **Antibacterial effects of the organic mercurial compounds, with special reference to their use as germicides . . .** *J. A. D. A.* **27**:276-283. Feb., 1940.

For this experiment, it was decided that pathogenic anaerobes should be chosen on the basis of their mode of attack on the body and the frequency with which they occur in nature. Clostridium tetani, Cl. septique, and Cl. welchii were chosen. For the proper approach to the problem, it was thought wise to secure some information concerning the actual use of these mercurials in the "cold sterilization" of instruments. For this, a questionnaire was sent to all class A and B dental schools in this country and to several dental clinics, and also to a representative group of individual dentists.

The conclusion reached was that none of the mercurials examined can be depended on to sterilize instruments when spore-forming organisms are present. Disinfection is different. None of the mercurials rendered the spores of Cl. tetani non-infectious after 24 hours contact, but the spores of Cl. septique and B. anthracis could be rendered non-infectious in certain instances. As regards their action on vegetative bacteria, it was found that one of the mercurials did not kill Staph. aureus in thirty minutes.

—*M. B.*

93—Grace, E. J.: **Anaerobic hemotoxin-producing streptococci in pyorrhea and lung abscess.** *J. A. D. A.* **27**:1481. Sept. 1940.

In cases of lung suppuration, it seems more logical that bacteria reach the lung by aspiration rather than through the blood stream. In the experience of the author, patients with lung abscess, especially of the chronic and putrid type, have invariably had dirty mouths, with marked evidence of pyorrhea. He has a series of ten cases of lung abscess in which the abscess cultures showed anaerobic hemotoxin - producing streptococci; in each of the ten cases, the mouth is pyorrheic and foul; cultures taken from the gums showed the same streptococci. In the opinion of Dr. Grace, this anaerobic hemotoxin-producing streptococcus is possibly the cause of pyorrhea, and the host of other organisms described are probably all formidable secondary invaders.

In chemotherapy, there are two excellent agents for dealing with this type of streptococcus; sulfanilamide, which has almost a specific effect, and zinc peroxide, which alters the medium from a destructive toxic anaerobic one to a relatively normal or harmless aerobic medium.

—*M. B.*

94—Hruska, A.: **Bakteriologische Untersuchung von Paradentaltaschen mit neuer Unter-suchungstechnik.** Bacteriological studies of paradental pockets with new technic. *Dtsch, Zahn, Mund, Kieferhlk.* **7**:9-13. Jan. 1940.

An attempt was made to obtain anaerobic bacteria from gingival pockets. This was unsuccessful. These attempts at obtaining material showed that samples of blood and purulent matter did not always yield desirable specimens, and that scaling and curettage of the pockets were necessary. The question of the possibility, that facultative aerobes could change to anaerobes was studied. The organisms had to be isolated in an airtight medium; the media used are described.

No distinction exists in possibility for infection from the periodontal space and the apical space. However, only from deep pockets of well-advanced paradentosis was there found bacteria containing material which suggested the possibility of focal infection. Paradental pockets should be removed by operation either directly before or after fixation of the teeth. The article is concluded with several case histories.

—*M. B.*

(d) Clinical Etiology

(Continued from 11(2):126. July, 1940)

434—Adloff, P.: **Die Paradentitis.** Paradentitis. *Med. Klin.* **35**:281. Mar. 3, 1939.

Dr. Adloff presents a review of the subject, paradentitis (he considers this a better name than paradentosis), with its etiology, diagnosis, and therapy. He considers local causative factors are more important than general endogenous factors, such as diabetes, vitamin C deficiency, etc. The role of the latter is too little understood, in its relationship to paradentitis, to be properly considered, except as any general disease weakens the resistance of the whole organism; and, since the paradentium is a "locus minoris resistantiae", it can the more easily be affected.

The primary cause of paradentitis is infection, which is non-specific; but its occurrence and its course are influenced by a variety of factors. He agrees with Haüpl that the distinction between paradentitis and paradentosis is incorrect. Clinically, the first phase is chronic gingivitis; this progresses to paradentitis marginalis, under the proper conditions, and finally to paradentitis profunda. Every case of gingivitis can develop into paradentitis, and every case of paradentitis must have developed from gingivitis. Symptoms, diagnosis, therapy, and prognosis of paradentitis are discussed. It is concluded that focal infection can develop from paradentitis, but the danger is not too great.

—*M. B.*

435—Euler: **Ueber die Entwicklung der Paradentose als Domestikationserscheinung und die dabei entstandene Kurve.** Development of paradentosis as a manifestation of domestication, and the resulting curves. *Paradentium.* **1**:1. 1940.

A review of paradentosis and caries, from the viewpoint of their historical development. In a study of skulls, dating from a few thousand years B. C. up to the present, the author used curves to show the occurrence of caries, parodontitis marg. progressiva, and parodontitis marg. superficialis. According to these curves, the former two diseases have risen sharply from about 1200 A. D., while the third has also risen but not as much. In reviewing the causes for this phenomenon, we cannot eliminate: climate, lack of sunshine, room for living, conditions of work, mode of living, etc., but in the foreground are the factors: eating habits and the changes which have occurred since the practically paradentosis-free time. However, since articles of food themselves were not so very different then from now, other factors must enter in; these are: combination, preparation, and physical constitution of the food commonly eaten.

—*M. B.*

436—Fuchs, H.: **Paradentose und Zahnarzt.** Paradentosis and the dentist. *D. Z. W.* **6**:89. 1940.

Two main symptoms of paradentosis are inflammation of the gingiva and atrophy of bone. Both of these occur in other conditions: the former in Moeller-Barlow's syndrome and scurvy, both due to vitamin C deficiency; and the latter in old age or loss of teeth. These symptoms in paradentosis must logically have the same causes as the same symptoms in other conditions, namely vitamin C deficiency and pre-senility; in some cases of paradentosis, the first symptom is more evident, and in some cases the second. Paradentosis always indicates a disturbance in metabolism.

The question of diet for paradentosis patients is discussed in which there are two main considerations: absorption of the natural vitamin C and remineralization of the jaw bone. The author elaborates on methods for taking care of these needs, and gives some clinical results; he includes sample menus of meals eaten by his patients. Local therapy, which must be given at the same time, is described with a short discussion of the dentist's responsibilities in regard to the problem of paradentosis.

—*M. B.*

437—Griebel: **Die Glossitis superficialis als Hypovitaminose und ihre Beziehungen zur Zungentuberkulose.** Glossitis superficialis as hypovitaminosis, and its relationship to tuberculosis of the tongue. *Klin. Wschr.* **14**:496. 1939.

Glossitis superficialis is an inflammatory disease of the mucosa of the tongue, which occasionally spreads to the mucosa of the cheeks and lips, and to the gingiva. On the dorsum of the tongue, especially on the sides and tip, are found scattered dark red spots, or streaks of deep red coloring. The papillae are inflamed and stand out sharply, often the tissue is swollen and shows marks of the teeth. The affliction is traceable to disturbance of the vitamins B^1 and C metabolism. A diagnostic point is that a sudden anorexia precedes the symptoms on the

tongue. When a general tuberculosis of the lung is present, a deficient supply of vitamin B can manifest itself in Moeller's glossitis; on the site of this condition on the tongue, tuberculosis of the tongue can ultimately develop. By means of the administration of large amounts of vitamins B^1 and C, both of these diseases can be successfully treated.

—*M. B.*

438—Hruska, A.: **Albaner und Paradentose.** Albania and paradentosis. *D. Z. W.* **43**(10):153-54. 1940.

Dr. Hruska considers that paradentosis should not always be conceived as being a pathologically correlated phenomenon. His own experiences, and especially his studies of various races all over the world, have taught him to regard paradentosis as a biological phenomenon, which is to be considered more or less as a racial characteristic, and hereditary. From this viewpoint, he states that paradentosis is a typical racial characteristic of the Albanian people. This people belongs to the Dinaric race of Near Eastern origin; they are a comparatively pure racial type. These Albanian people show only slight tendency toward abrasion, but an early loosening and loss of their teeth. They are compared with the mixed group of peoples classified as Yugoslavs; these latter are a direct contrast, with a great deal of abrasion and no widespread paradentosis. The author considers the Albanians a newfound field for the study of paradentosis, and advises that they be studied from an anthropologic-odontological standpoint.

—*M. B.*

439—Jacquard, R.: **Die Vererbung und die Theorie Weismanns in Zusammenhang mit den Paradentopathien.** Heredity and Weismann's theory in relation to paradental diseases. *Paradentium.* **12**(2,3):17-22. 1940.

Dr. Jacquard gives a brief summary of the important work of Weismann, dealing with the germ plasm; according to his theory, the germ plasm is immutable; no external changes in the rest of the body could produce any change in the germ plasm, and only through direct changes in the germ plasm itself could any change in the species be brought about. On the other hand, we know now that, in each individual, the environment may influence both the appearance and development of these organic determinants (potentialities), inherent in the germ plasm. It is therefore possible in some measure to modify the factors presented by the paradentium which is idiopathically enfeebled. We should pay more attention to the condition of the paradentium in people who marry; an individual with a good general constitution should not marry one with a poor constitution, nor should an individual with a tendency toward severe paradental disease marry another with the same tendency. One of our tasks should be to study the geographic distribution of paradental diseases, in order that we may assess the etiologic value of the factors, heredity and environment, in them.

—*M. B.*

440—Kramer: **Der Verlust der Nahrungsmittel, insoderheit der Kartoffel, an C-Vitamin durch langes Warmhalten.** Loss in vitamin C of food, especially potatoes, through long heating. *Paradentium.* **1**:5. 1940.

An increasing percentage of people cannot obtain their minimum vitamin C requirement from food with the prevailing cooking methods, especially those whose food is cooked in large kitchens. Only when food, under its ever-changing conditions of consumption, is studied with the control of animal experiments, can we get a true picture of the vitamin C content of the food concerned. A change in cooking methods is absolutely necessary. Potatoes, which are the main source of vitamins for most of the people, when prepared unpeeled and steamed, lose only small amounts of their vitamin C content. With a minimum consumption of 300-400 gm. the minimum vitamin requirement can be secured from the native food, the potato. Long heating makes vegetables and potatoes worthless in their vitamin C content. It is also probable that vitamins B_1 and C have a synergistic action.

—*M. B.*

441—Kutzleb, H. J.: **Ueber die Aetiologie und die Behandlung der "Stomatitis ulcerosa sexualis."** The etiology and treatment of "stomatitis ulcerosa sexualis." *Z. Rund.* **49**(11):351-57. 1940.

On an etiologic basis, the author divides stomatitis ulcerosa into seven types: infectiosa, hematopoetica, intestinalis, allergica,

toxica (or medicamentosa), hypovitaminotica, and incretorica, the last-named being caused by disturbances of the endocrines, which, if due to disturbance of the sex hormones, can be called stomatitis ulcerosa sexualis. Before a particular case can be definitely established as due to hormonal disturbance, all the facts and symptoms of that case must be thoroughly investigated, and all other possible causes eliminated.

The follicular hormone has been shown to have three actions:

1. Vaso-dilatatory action.
2. Infection-preventing action.
3. Action on the epithelium, especially on the genital mucosa.

In cases of hypovitaminosis and disturbance of the sex hormones, disorders of both the vaginal and oral mucosa have been found. Anemic-atrophic form of gingivitis, together with vaginitis senilis atrophicans, are found in cases of hypovitaminosis; however, positive oral findings do not always concur with an apparent vaginal symptom, nor vice versa. In stomatitis ulcerosa, in both men and women, good curative results can be obtained by the administration of follicular hormone, Progynon-DH, or of a substitute preparation.

—*M. B.*

442—Lucyk, M. E.: **Mouth breathing gingivitis.** *D. Students Mag.* Apr. 1940.

Mouth breathing gingivitis is an inflammatory reaction of the gingival tissues to injuries received through physico-chemical action—physically, because of dryness, and chemically due to lack of cleansing by flow of saliva and the rubbing of the lips. There is marked inflammation of the gingival tissues about the labial surfaces of the upper or lower jaws, or both. The gingivae are red and swollen, with round blunt contour, bleeding at the slightest touch, stippling is absent, producing a shiny surface. Gingival pockets are present; the lips are usually dry and cracked. The patient may have frequent colds.

The patient may be unaware of his mouth breathing habit, and it may take place only during sleep. The habit is chiefly caused through infection of the nasal lymphoid tissues and nasal passages, or by some bony obstruction in the nose. The habit itself must be corrected through persistent effort on the part of the patient; lip exercises are beneficial. Mouth screens can be used at night. Local periodontal treatment clears up the local irritation and improves gingival circulation. The condition in the nose must be corrected to remove the primary cause.

—*M. B.*

443—Kronfeld, R. and Weinmann, J.: **Traumatic changes in periodontal tissues of deciduous teeth.** *J. D. Res.* **19**:441-454. Oct. 1940.

Traumatic injuries found in the supporting tissues of 6 deciduous dentitions, ranging in age from 9 months to 4½ years, are reported in this study. The least severe of these injuries consist of a compression, ischemia, and hyalinization of periodontal connective tissue. The most severe ones caused crushing and necrosis of periodontal tissues and the formation of hemorrhagic cysts. In most instances, injuries were followed by undermining bone resorption and by evidence of tissue repair.

The etiology of these injuries is probably the weakness of periodontal membrane of erupting and newly erupted teeth. During these periods, periodontal tissues are not yet fully equipped to withstand mastication stress; such a work is comparable to one whose periodontal membrane has been weakened by resorption of alveolar wall. In somewhat older jaws, weakening of periodontal support caused by shedding is an additional factor in reducing resistance of periodontal support. Clinical manifestation of such traumatic injuries in deciduous dentition may be slight soreness of erupting teeth, and later on, perhaps, ankylosis between teeth and bone by reparative formation of bone and cementum.

—*M. B.*

444—Martiny: **Die Enstehung und Bekaempfung der Konkrementbildungen an den Zaehnen und in den Speicheldruesengaengen.** Formation and control of calculus on the teeth and in salivary ducts. *Paradentium.* **12**(2, 3):36. 1940.

The organic "scaffolding" of calculus is mainly built up by actinomyces, with the help of leptothrix and other mouth bacteria; without actinomyces, no calculus is formed. Through their proteolytic power, they produce amino acids, which, in spite of their acid character, give an alkaline reaction;

this alkaline medium causes the precipitation of carbonate and phosphate from the saliva. Actinomyces live as well under aerobic as under anaerobic conditions. The varying deposition of calculus in each individual occurs according to the varying biological conditions of the oral cavity with regard to the development of calculus-forming bacteria. In cases of a high bacterial content, pathologic conditions may develop, not only of the paradentium, of the mouth, and of the respiratory tract, but also in the form of a skin infection.

Dr. Martiny recommends a complete change in our usual mouth hygiene methods, after the calculus is cleared up. He denounces all calcium carbonate or tricalcium phosphate and soap tooth pastes, and recommends a definite acidity for a toothpaste, to stimulate the saliva and to make it more alkaline.

—*M. B.*

445—Meyer, G.: **Askobinsauere in Konserven.** Ascorbic acid in preserves. *Schw. monat. f. Zahnbl.* **50**:902. Oct. 1940. Abstract.

In the laboratory of a preserve factory in Lenzburg, a series of titration experiments were conducted, according to Tillman's method, on raw fruits and vegetables, and also on the finished preserves. Desirable animal studies could not be carried out, so that certain sources of error could not be entirely eliminated. The evaluation of findings must therefore be made with certain reservations. Various types of preserves were studied. The results showed that, preserves of native fruits and vegetables, are fair to very good sources of vitamin C. In wartimes, during which the population is made to lay-in stores of food, this knowledge is important.

—*M. B.*

446—Miller, S. C.: **Periodontal problems in inefficient mastication.** *J. Sec. Dist. D. Soc.* **25**(6):399-404. 1940.

The article deals with the problem of mastication and occlusion, as related to periodontal, and general oral health. There are essentially two ways in which traumatic occlusion may be produced: first, by excessive strain on the teeth themselves, and second, by weakening of the supporting structures. The first may be produced by insufficient wear of the occlusal surfaces, placement of restorations, or through biting habits; the second may be induced by systemic disease or inherent bone weakness.

The various problems in malocclusion and their orthodontic treatment are briefly discussed. The position of the teeth in the arch is not static, but depends on many factors—continuity of the arch, occlusal function, tongue and lip pressure, and muscle tonus. When the periodontal membrane is intact and the same amount of elastic holding force is exerted in all directions, the tooth is in equilibrium. However, when a portion of the periodontal membrane is lost due to disease, surgery, alveolar recession, etc., there is unequal pull on the remaining fibers, and the slightest degree of occlusal pressure will induce movement of the tooth out of position, causing migratory periodontal disease. The diagnostician should keep in mind jointly the elimination of focal infection and the restoration of function of the teeth; it is impossible to completely eliminate periodontal disease for any length of time without careful attention to the restoration of function.

—*M. B.*

447—Morgenroth, K.: **Traumatische Schaedigungen des Zahnfleischrandes.** Traumatic injuries of the gum margins. *Dtsch. Zahn, Mund, Kieferhlk.* **7**:13-59. Jan. 1940.

From among the great number of etiologic factors concerned in paradentopathy, the author elects for consideration, direct mechanical influences which act upon the gums; he used young dogs as experimental animals. He considers the gingival epithelium to be histologically connected with the enamel at the gingival margin. The gingival margin is normally protected by the convexity of the teeth, and in the interdental spaces, by the contact point.

Dental operative procedures often cause traumatic damage to the gingiva; of these, the worst is surgical cutting of the gum margin advocated by many authors in apicoectomy. According to W. Meyer, who has studied this on a histologic basis, it is only indicated where a paradontitis marginalis chronica progressiva exists; Meyer's studies are then cited. It has also been shown that separation of the gingival margin from the enamel, with quite similar results, can occur from the use of orthodontic liga-

tures, and even from the rubber dam; a discussion of the proper and improper ligatures, with the evil consequences of the latter, follows. Finally, the possible damage caused by the margins of dentures, and denture clasps, is evaluated.

—*M. B.*

448—Schwarz, M.: **Die Beziehungen der Kieferorthopaedie zu den Paradentalerkrankungen.** The relationship of orthodontia to paradental diseases. *Dtsch. Zahn, Mund, und Kieferhlk.* **7**:(2):82-89. 1940.

It is now well-established that paradentosis is an independent disease group with its own predisposition. This predisposition is of varied nature; we are familiar with its relationship to hormonal disturbances, metabolic disorders and disturbances of diet. The paradentium has been cited as a good indicator for such disturbances of general health, but it is known that external factors are very important in determining the severity and course of the disease. In this article, Dr. Schwarz considers to what extent orthodontia can act as one of these external factors in the production of paradentosis. According to him, two possible injuries can result:

1. Through orthodontic intervention, paradentosis might be started, since each orthodontic measure means overstrain and this can cause paradentosis.
2. Orthodontic measures could cause resorption.

The author discusses these points, citing the views of various men in the field; he considers these possible means of damaging the paradentium as being clinically unfounded, and illustrates with several cases of his own.

—*M. B.*

449—Sigmund, H.: **Die Beziehungen der Kieferorthopaedie zu den Paradentalerkrankungen vom Standpunkt der Pathologie.** The relations of orthodontia from the standpoint of pathology. *Dtsch. Zahn, Mund, und Kieferhlk.* **7**(2):65-82. 1940.

The paradental conditions in anomalies of position are in conformity with prevailing functional conditions as in cases of overstrain and in lack of any strain. They develop into paradental disease, especially loosening of the teeth and atrophy of the alveoli, *only* when an individual constitutional tendency for paradentosis exists or is acquired.

Through orthodontic measures, there is affected a thorough building up of the functional paradentium, which finally develops into a new equilibrium in the entire dentition. Inflammatory symptoms in the soft tissues, which often appear during orthodontic treatment, due to mechanical irritation of the mucosa, are to be evaluated only as ordinary marginal gingivitis; it develops symptoms of paradentitis progressiva profunda, with destruction of alveolus, only where the reconstruction material of the body is insufficient. Further study and research for this complex problem is needed.

—*M. B.*

450—Sorrin, Sidney: **Chronic desquamative gingivitis.** *J. A. D. A.* **27**(2):250-257. Feb. 1940.

Author's summary and conclusions:

"1. Both men and women may suffer with this condition.
2. A most characteristic sign is desquamation of the epithelial tissues, which leaves a raw bleeding surface.
3. The disease is confined to the gingival tissues and rarely involves bone.
4. It may be found on labial, buccal and lingual gingivae. However, it does not appear so often on the lingual gingivae as it does in other regions.
5. Pain may be present.
6. A fetid odor is usually present.
7. Careful local or systemic check-up has revealed no condition which might lead one to suggest at this time any one etiologic factor present in all cases.
8. Beechwood creosote in iodine (Adair treatment) seemed to help in the last two cases treated.
9. At the present time I am experimenting with prontylin (sulfanilamide), applying this agent topically to the affected parts by making a paste with water."

"It is strange that other parts of the body do not present similar changes in chronic desquamative gingivitis. More study on the saliva, blood and urine might reveal the factors in the disease. The review has been

presented with the hope that some one, somewhere, is successfully treating these cases and has found one etiologic factor in the condition and has also found an agent which will cure it."*

451—Thoma, K. H. and Goldman, H. M.: **Wandering and extrusion of teeth, and pocket formation, in paradentosis.**

Paradentosis, defined by the authors as diffuse atrophy of supporting structures of the teeth, allows the teeth to migrate and become loose. Only single-rooted teeth are affected (multi-rooted ones offer sufficient resistance), and they may show mesial and distal, as well as labial movement. In early stages, no pockets are present and gingivae are not affected.

First, the bone is resorbed in plane vertical to the alveolar margin by osteolysis and vascular resorption; this causes detachment of the principal fibers of the peridental membrane from the alveolar bone, their function is lost, and they also become detached from the tooth. Pocket formation is of vertical type because the bone is resorbed in the vertical plane, and as a result, the peridental membrane becomes wider. At the same time, the epithelial proliferation progresses rootwise along the cementum; this becomes detached from the cementum, forming a pocket, which becomes infected. Pathologic wandering, according to the authors' interpretation is due to the pressure from the proliferation of connective tissue replacing the resorbed bone; proliferation completely around the tooth causes extrusion. Occlusion influences direction of migration and the site of the pocket formation.

—*M. B.*

452—Wannenmacher, E.: **Ein Beitrag zur Aetiologie und Therapie der Paradentopathien.** Contribution to the etiology and therapy of paradental diseases. *Schw. Mschr. f. Zahnhlk.* **50**(4):329-42. 1940.

The formation of the paradental pocket is dependent on the following conditions: extension of the epithelial gap, itself dependent on the quality of the epithelium and on trauma; intensity of foreign invasive material, in the form of bacteria; resistance of the local tissue and of the whole organism. The clinical picture of paradental disease is exceptionally variable; it is therefore very important to synthesize all clinical details and to fix the particular form of the disease. Paradental diseases are divided into two general classes; paradentoses, in which atrophy is the main feature, with little or no inflammation; and paradentitis marginalis chronica progressiva, in which inflammatory signs are dominant.

The inflammatory type of paradental disease is found in every age group. Inflammatory changes of the gingiva, paradental pocket formation, suppuration, bone resorption, and formation of concretions, in varying degrees, are the most striking symptoms of paradentitis marginalis chronica progressiva. The author then presents a discussion of the various factors concerned in producing this disease, such as occlusion, function of the jaws, hormonal factors, etc. Besides these endogenous factors, various external factors are involved, which act especially on the interdental papillae.

Dr. Wannenmacher's local therapy consists, first, of curettage, with various instruments, and addition of chemicals for removal of concretions; then, the use of astringent solutions. The use of a postoperative pack to relieve pain is also very valuable.

—*M. B.*

453—Watsky, S.: **The knife and fork, primary causes of periodontoclasia, dental caries, and malocclusion.** *N. Y. J. Den.* **10**:232-34. 1940.

The use of the knife and fork, as implements of prehension and incision, deprives the anterior teeth of their natural function; added to this, modern soft foods give very little exercise to the posterior teeth in chewing and grinding. Abrasive elements in the food are usually thought necessary for the attrition of cuspal planes and a resultant end-to-end bite; but the author considers that as long as the knife and fork are used, no amount of abrasion of the posterior teeth, or nutritional perfection, will compensate for the lack of attrition through continuous free exercise of the incisive and prehensile functions of the edges of the incisors and cuspids. The overbite, unreduced by wear directly on the edges of the anteriors, restricts the excursions of the mandible to

*See *J. Periodont.* 12(1):53. Jan. 1941. Abstr. (g)589.

short arcs of extremely limited radii. The unreduced cusp height and locked interdigitation result in trauma and pathology of the periodontal membranes, and in malocclusion; further, the locked occlusion encourages food stagnation and resulting caries.

—*M. B.*

454—Zemsky, J. L.: **Endocrine disturbance and periodontal disease.** *D. Outlook.* **27**:541-43. Dec. 1940.

Present available material does not permit definite conclusions as to the existence or non-existence of a relationship between endocrine dysfunction and periodontal disturbances. The case reported by Dr. Zemsky indicates such a relationship. The patient was tentatively diagnosed, on the basis of roentgenographic and clinical findings, as a case of diffuse alveolar atrophy. To confirm this, a metabolic test was necessary, to determine the specific dynamic action of protein, which is pathognomonic of this type of periodontal disturbance. Weinmann found that patients with alveolar bone atrophy showed less than 20% increase in basal metabolic rate, after injecting 100-150 grm. steak, while the normal increase is 20% to 40%. Weinmann says this can be interpreted as due to hypofunction of the anterior lobe of the pituitary, or a disturbance of the vegetative nervous system. Therapy instituted in this case was surgical, medicinal, and dietetic; since it was a case in which the endocrine disfunction caused periodontal disturbance, correction of systemic derangement resulted in cure of local lesion.

—*M. B.*

* * *

CORRECTION

Abstract (f)528-J. *Periodont.* 2(1):36. January 1941. Author of original article and abstract, Frank E. Beube. The abstract read in part ". . . due almost entirely to its strong alkalinity, and are largely dependent on the sulfhydryl and sulphid ions . . . " It should read ". . . due entirely to its strong alkalinity and independent of its content of sulfhydryl and sulphid ions . . . "—*Ed.*

BOOK REVIEWS

Twelve Periodontal Studies. By Harold Keith Box, D. D. S., F. A. A. P., Toronto, Canada. The University of Toronto Press, 1940, 251 pages.

The "Studies" presented in this latest book by Box were delivered as separate lectures; some of them have been published elsewhere in part at least. Each study is complete in itself and is prefaced by an outline which indicates quite definitely the matter discussed in it. The author's researches in periodontal pathology have gone hand in hand with his teaching and practice of periodontia for a quarter of a century and he has applied this rich experience in the present studies.

Box contrasts the physiologic wear which occurs evenly on all the teeth in primitive man, with the absence of such wear in the modern dentition. As a consequence of retarded occlusal wear in early life we find interfering cusps and overloading in the adult especially where the periodontal structures are poorly developed. Under this latter condition it is recommended that some artificial abrasive adjunct be used, under the supervision of the dentist.

The physiologic strengthening of the periodontium under increased stress is to be expected only when such overloading is mainly axial. When these stresses act horizontally they must be sustained more or less regionally by the periodontium and the reaction is one of injury instead of benefit.

The pathology incident to lack of function is described as a loss of cellularity with a striking change in the architecture of the supporting fibrous structures in general; and these atrophic changes Box calls "adaptive fibropenia." We are indebted to Box for the term "rarefying pericementitis fibrosa" also, and for "periodontium" to designate all the supporting structures of the teeth as a group unit.

One is impressed from the outset with the fact that this work is not orthodox, that Box draws his own conclusions unbiased by concepts generally held. The following paragraphs are characteristic.

> The mucin content of saliva has been suggested by some writers as a local factor which predisposes to dental caries. . . . It must be pointed out, however, that the oral vestibule, notably the division of the oral cavity prone to cervical caries, is under the dominance of parotid saliva, a thin secretion devoid of mucin.
>
> The viewpoint has frequently been expressed that the exudate from the gingival crevice is acid in reaction in inflammatory conditions. This opinion has not been confirmed by our investigations. On the contrary, the findings tend to show a slight increase in alkaline reaction.

Box's unconventionality and independence of thought are demonstrated in Study VII, "Periodontal Therapeutics," and the two succeeding chapters on case management. While consideration is given to all the generally accepted procedures in the handling of periodontal cases, including examination by every available means and not omitting due attention to cleansing, polishing and home care, Box's book devotes more space and attention to the so-called Dunlop treatment than to all other therapeutic measures.

It was evidently some years after the publication of his *Treatment of the Periodontal Pocket* that Box became interested in the Dunlop method. It would seem from articles which have appeared of late in the *Journal of the Canadian Dental Association*, notably an editorial in the July, 1940 issue (pp. 351-53) that its use is more common in Canada than in the United States. In an article, "The Simplifying of Pyorrhea Treatment," in the *D. Items Interest* (62(6): 538-39, June, 1940), Gottlieb re-affirms his belief in oxygen as a therapeutic agent.

The drugs and materials employed in the Dunlop treatment have been considered by many of us to be more or less in the proprietary class, and the entire method empirical, but one cannot dismiss lightly these recommendations by responsible and distinguished dentists. To say the least, Box makes out an excellent case for "Dunlop treatment."

In studies IX and XII there are some interesting and unusual observations on immunity to caries and on periodontal conditions and caries.

Throughout the book original and stimulating suggestions can be found.

Like Box's former *Studies in Periodontal Pathology* (1924), *Treatment of the Periodontal Pocket* (1928), and others, the present small volume is from the Press of the University of Toronto in which university Box holds a professorship. The unglazed paper and broad clear type make reading the easier, but one misses the fine photomicrographs which were numerous in his earlier works.

—*Celia Rich*

Histo-pathology of the Teeth and their Surrounding Structures by Rudolph Kronfeld, B. S., M. D., D. D. S., Professor of Dental Histology and Pathology in the Chicago College of Dental Surgery, School of Dentistry, Loyola University, Chicago, Illinois; worker of the Foundation for Dental Research of the Chicago College of Dental Surgery; Fellow of the American Academy of Periodontology; Former Assistant at the Dental Institute of the University of Vienna, Austria. Second Edition, Thoroughly Revised. Philadelphia, Lea & Febiger. Illustrated with 438 Engravings, 504 pages. 1939.

The first edition of Kronfeld's Histopathology of the Teeth and their Surrounding Structures published in 1933 was extremely well received. It was reviewed in *J. Periodont.* 8(1). Jan. 1937.

The second edition which appeared hardly a year before his death lends additional lustre to the name of the brilliant young scientist-author.

Dr. Kronfeld was very proud of this, his last work, and he himself thought it a great improvement over the first edition.

In it he has omitted certain chapters, notably that on "Histology and Physiology of the Dental Tissues," since the contents of this chapter have been elaborated and included in another of his books *(Dental Histology and Comparative Dental Anatomy)*. The chapter on Histological Technic was dropped because it too is available elsewhere.

On the other hand several subjects and chapters have been added and some fifty illustrations.

Among new subjects treated are physiology and pathology of tooth calcification; erosion; bacteriology of caries, pulpitis and periodontitis; focal infection; healing of extraction wounds, Vincent's infection, and some of the diseases of the oral mucosa, including tumors of the oral cavity.

The general principles and important problems of calcium metabolism and calcification are clearly set forth. It is worthy of emphasis and reiteration that although developing teeth are very sensitive to variable calcium metabolism this is not true of adult fully erupted teeth. In bone there is a continuous process of calcification and calcium withdrawal, but in teeth calcification only occurs. It has never been demonstrated that minerals can be withdrawn from teeth, indeed the calcium content of teeth is stable even when, due to some systemic disturbance, large amounts are withdrawn from the skeleton. In x-rays of hyperthyroid cases the teeth stand out sharply against the poorly calcified jaws.

An interesting series of pictures shows the amazing destruction of the supporting structures in a diabetic in 18 months time.

The presence of calcifications within the pulp is almost universal even where the x-ray does not disclose them and there is no justification for interpreting such formations as infective; nor is the mere presence of bacteria in pulps of intact teeth, with or without pulp stones, to be so considered unless pulps show other evidences of inflammation.

A nice distinction is drawn between caries of vital and non-vital teeth and the entire discussion of the caries problem is adequately handled.

The section on diseases of the periodontal tissues so ably presented in the first edition has been brought up to date. In the excellent classification table the nomenclature used by Box and that of Becks have been added to the author's and that adopted by the F. D. I.

In the discussion of granulation tissue we read:

> Its formation is a defensive or reparative reaction . . . in many instances granulation tissue initiates the healing of an infection or wound.

It is young highly vascular tissue.

The paragraphs and the photomicrographs of the healing of extraction wounds form a worthwhile addition to the volume as does also the new chapter on Vincent's Infection and Common Diseases of the Oral Mucosa.

Of focal infection Kronfeld says,

> The existence of a focus does not *per se* constitute

actual focal infecion. The bacteria may remain confined within the focus . . . .

There is potential danger however whereever pathogenic organisms have gained a foothold in the tissues.

The ultimate solution of the focal infection problem lies with the dentist. This solution can be stated with one word: prevention.

The second edition of *Histo-pathology of the Teeth and their Surrounding Structures* has been revised throughout both, as to phraseology and subject matter and the sequence of the chapters improved.

Kronfeld has again demonstrated his ability to touch the high spots of a subject and throw them into clear relief.

The book is a beautifully executed volume as to paper, print, and illustrations.

The additional bibliographical data alone make it worthwhile. It contains comprehensive and exhaustive references through 1938.

—*Celia Rich.*

El Paradencio-Su Patologia y Tratamiento (The Pathology and Treatment of Parodontal Disease). By Dr. Francisco M. Pucci; published by J. Garcia Morales, Montevideo, Uraguay. 1940.

From the wide range of the material assembled in this book and from its unusual size and the great number of illustrations, this worthy contribution to dental literature might well be termed an atlas-encyclopedia of periodontology and allied subjects.

The book-covers, approximately 8x12 inches, contain between them 683 fully utilized pages, including 762 illustrations, in addition to colored photographs, and full page or folded illustrations or charts. The type is unusually clear and makes easy reading. The numerous photographs and microphotographs are very well reproduced generally on paper especially adapted for such material. The volume consists of two parts, the first, under the subject of "Periodontal Biology and Pathology," the second, "The Clinical Aspects of Periodontal Disease," including diagnosis, prognosis, treatment, prevention, etc.

The author takes full advantage of the contemporary tendency to enlarge considerably most photographs, x-rays and microphotographs, as well as diagrams. This affords reproduction of greater detail and ease of perusal. He has evidently gone to no end of effort in his tremendous undertaking, to make the book "a bigger and better one," as we Americans would say. Dr. Pucci has drawn liberally upon literary and illustrative material from many authors, for which due acknowledgment is made. However, he shows evidence of vast personal experience and study in the subject of periodontology, and, accordingly, his opinions and methods of procedure are presented, side by side, with those of a series of contemporaries. The work of numerous members of our Academy is well represented in the volume. Dr. Pucci has obviously done a signal service for the South American and other Spanish-speaking dentists in accumulating and presenting in their own language, in a masterful way, so much periodontal literature drawn from English, German, and French textbooks and periodicals. He is to be congratulated for this splendid contribution to the science of periodontology.

—*Isador Hirschfeld.*

A Text-Book of Oral Pathology. By Russell W. Bunting, D. D. S., D. D. Sc., Professor of Dental Histology and Pathology, School of Dentistry, University of Michigan, Ann Arbor, Michigan, and Thomas J. Hill, D. D. S., Professor of Clinical Oral Pathology and Therapeutics, Western Reserve University, Cleveland, Ohio. Second edition, thoroughly revised. Philadelphia, Lea & Febiger, 460 pp. 316 illustrations. 1940.

This second edition of *A Text-Book of Oral Pathology* by the Dean of one of our leading dental schools and associated authors, Dr. Thomas J. Hill, Dean B. E. Lischer, and Samuel W. Chase, Ph. D., is generously and well illustrated. The clear style of writing is characteristic of Dr. Bunting and no dental student could possibly find this textbook a dull one. The selection and arrangement of the numerous illustrations add much interest to the text. For the dental practitioner the book might be classed as easy reading.

Dr. Bunting's chief professional life interest has been research in dental caries and two chapters are devoted to this subject. There are seventeen chapters in all. Abrasion and erosion are given an entire chapter which is especially enlightening. Pathology of the supporting tissues of the teeth is well presented in three chapters. It is to be regretted that the author of these latter chapters should have found it necessary to con-

tinue to use the term pyorrhea alveolaris which he admits is a misnomer. This is particularly undesirable in a text-book when so many dental writers are making a serious effort to improve our tendency to be careless in the use of terms. Dr. Bunting states:

> Generically it [pyorrhea alveolaris] indicates a flow of pus from the alveolus, inferring that a purulent exudate is a characteristic feature of the disease. This is not true. In the great majority of cases the predominant types of infection associated with the disease are apurulent in character and there is no purulent exudate. When used to describe the disease it is necessary, therefore, to speak of purulent and apurulent types of pyorrhea, which is indeed an anomaly.

This might be described as "confusion worse confounded."

The importance of prevention is emphasized throughout the book, references are numerous but a proportionately small number of them are to writings after 1935.

A Text-Book of Oral Pathology can be recommended as enjoyable reading to even a disinterested dentist who may in this way pleasantly refresh or stimulate his interest in the various dental and periodontal diseases with all of which he should be familiar.

—*G. R. S.*

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